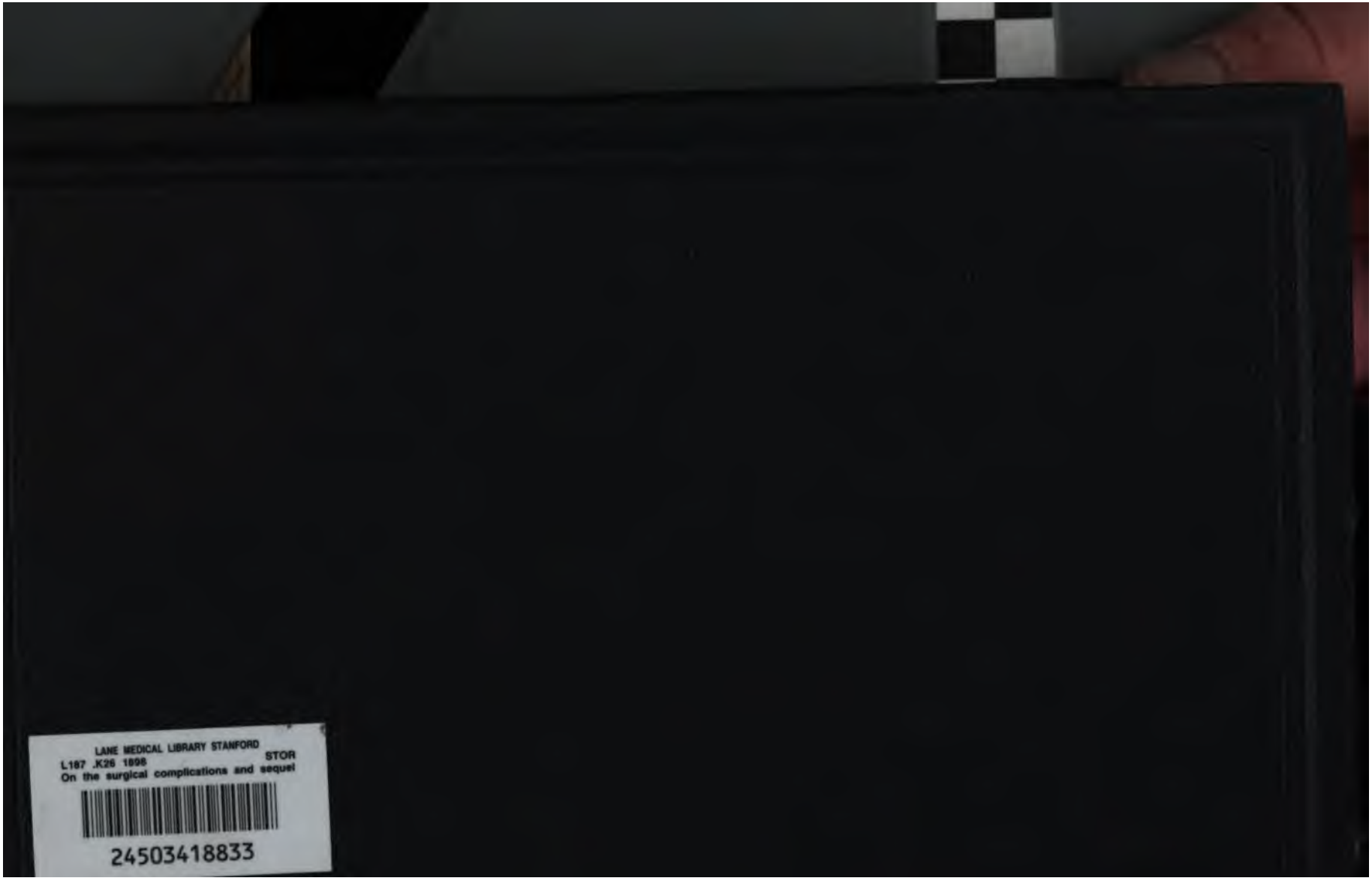

THE

SURGICAL COMPLICATIONS AND SEQUELS OF TYPHOID FEVER

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BASED UPON TABLES OF 1700 CASES

COMPILED BY THE AUTHOR AND BY

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AND AS AN APPENDIX

THE TONER LECTURE, No. V.

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LIST OF ILLUSTRATIONS.

TONER LECTURE.

ERRATA.

Page 40, line 3, for *once* read *one*.

Page 74, line 8, for *anus* read *arms*; line 9, for *20* cases read *21*; line 13, for *151* read *147*; line 14, dele *and anus*.

Page 183, line 7, for *362* read *352*.

SURGICAL COMPLICATIONS AND SEQUELS

OF

TYPHOID FEVER.

CHAPTER I.

INTRODUCTION.

THE present monograph had its origin in two lectures. On February 17, 1876, I delivered the fifth Toner Lecture "On the Surgical Complications and Sequels of the Continued Fevers," which was published by the Smithsonian Institution in March, 1877. On June 9, 1896, I delivered the Shattuck Lecture, before the Massachusetts Medical Society. In casting about for a subject for the latter lecture, it occurred to me that it would be just twenty years since my Toner Lecture, and that the literature accumulated on the subject during that period would well warrant a sequel, a sort of "*Vingt ans après.*" Accordingly, I asked Dr. Thompson S. Westcott to undertake the tabulation of the cases recorded since 1876. They amount to over 900 cases.

Not only did he tabulate the cases, but also accurately analyzed their results. That he has done his task with completeness and accuracy is evident in every chapter. It was evident that a single lecture

would not suffice for the discussion of so extensive a mass of material, and hence I restricted the Shattuck Lecture to "Gangrene as a Complication and Sequel of the Continued Fevers, Especially of Typhoid." (Trans. Mass. Med. Soc., 1896, and Boston Med. and Surg. Jour., July 2 and 9, 1896.) This Lecture I have made Chapter III in the present book, though much altered to fit it for its new place.

I determined, therefore, at my earliest leisure, to take up the various other complications and sequels of typhoid, and so make as complete a monograph as possible. I was the more disposed to do so because, though individual complications or sequels had been considered by various authors, no one since my previous attempt had treated the subject as a whole.

I was also the more desirous of doing so because of the fact that the bacillus of typhoid had been discovered by Eberth in 1880, and hence our views of the pathology of these surgical lesions had undergone a great and necessary change. Yet even now almost every chapter shows how deficient we are in precise knowledge of the exact nature of these lesions. Every case hereafter reported should be completed, if possible, by a bacteriological examination. Negative evidence is as important as positive.

How large a rôle the complications and sequels of typhoid play is well shown by the statement of Hölscher (Münch. med. Wochen., 1891, No. iv) that of the 2000 fatal cases there tabulated, only 24 per cent. died as a result of the typhoid infection *per se*, and 76 per cent. from the various medical and surgical complications and sequels.

The Toner Lecture and the present book together cover 1700 cases, and practically include nearly all the cases recorded in the last fifty years. The table ends with the literature of 1895, except a few important cases which have been reported after the table was completed and analyzed. The cases of Perforation of the Intestine (Chap. XV) and those of Perforation of the Gall-bladder (Chap. XVI) have been brought down to January, 1898.

In the analysis of results (as to age, sex, location, etc.), unless otherwise stated the figures combine the results of the two series of cases of 1876 and 1896. Single cases not seldom reappear in several chapters, since a case of gangrene might also suffer from a periostitis and a parotitis, and require to be considered under each heading. Such multiple lesions are quite characteristic of typhoid.

The diagnosis in a moderate number of cases is somewhat doubtful, but I have not felt at liberty to change it. The number of errors from this source, as well as those arising from the indefiniteness and meagerness of some of the histories, is not so great as to impair the substantial accuracy of the analytical results, especially for clinical purposes.

In the present book I have considered not only the six complications and sequels treated in the Toner Lecture, but I have added a formal chapter on the Pathology of the Surgical Affections of Typhoid, and also twelve chapters on other surgical affections of particular organs and regions. The surgical treatment of intestinal perforation did not exist in 1876. One of the principal additional chapters, from the practical standpoint, is upon this sub-

ject. As this is so vitally important I have included a table of all the cases thus far reported. In connection with this chapter I wish to call especial attention to the "conclusion" No. 10, formulated on page 318, that the *essential mortality* is not the mortality of the whole series (80.7 per cent.), but the mortality of those cases operated upon within the first twenty-four hours (69.4 per cent.), and that this will be further reduced when prompt surgical interference is the rule. In the chapter on Typhoid Affections of the Gall-bladder I have called attention to the propriety of immediate operation in peritonitis arising from similar perforations of the gall-bladder —a surgical chapter just opening. Four such cases have been operated upon with three recoveries.

I have slightly changed the title of the present work both to distinguish it from my Toner Lecture and because nearly all of the cases in the second series are the surgical results of typhoid fever alone. In the first series of 785 cases (excluding 352 of parotitis following typhus), 252 cases followed typhoid, 119 followed typhus, and 62 followed other continued fevers. In the second series, 887 followed typhoid, only 23 cases followed typhus, while 4 followed other continued fevers. No better or more cheering evidence of the improved sanitation of the civilized world could be given than this palpable diminution in the ravages of typhus fever. At the same time it is to be noted that these figures are to some extent deceptive. The unfortunate habit, especially of the Germans, of designating typhoid fever as "typhus abdominalis," and (from the far greater frequency of typhoid) the not infrequent use

of the word "typhus" alone, without the qualifying adjective, when typhoid was really the disease, makes the number of cases of typhus somewhat larger than it really is. Happily in the second series this is much less frequent. If a true typhus bacillus should be discovered, how will our German friends discriminate between the two? What endless confusion will arise!—a confusion so easily avoided if the adjective "typhoid," or "enteric," were now used to designate the so-called "typhus abdominalis."

The original tables on which my Toner Lecture was based unfortunately were not preserved. Hence in some of the analyses the later table alone has been available for points not covered by the earlier lecture. Where this is the case it is so stated. At first I had intended to publish also the tables and the bibliography in full, but this would have so increased the bulk and the cost of the book that I reluctantly relinquished the idea. Instead of this I have given copious references.

The ophthalmic lesions after typhoid are such a special class that I have asked my friend, Prof. George E. de Schweinitz, to write that chapter. The thoroughness and completeness of this chapter, like all his work, is very noteworthy. Both to him and to Dr. Westcott I desire to express my earnest thanks. I should also mention Profs. William H. Welch, William Osler, W. M. L. Coplin, and D. Braden Kyle, to whom I am indebted for valuable suggestions and assistance. I also wish to acknowledge the courtesy of a number of gentlemen who have sent me notes of unpublished cases.

In order to make the work as complete as pos-

sible I have added, in a *Postscript* (page 321), a number of cases of importance which have appeared as the book has been passing through the press. They concern Chapters II, VIII, XVI, and XVII.

I would be wanting both in courtesy as well as in gratitude did I not take this opportunity to express my obligation to the gentlemen in charge of the library of the Surgeon General's Office, U. S. A., for placing at my disposal both twenty years ago and now the treasures of that unrivalled collection. Without that, the Index Catalogue and the Index Medicus, it would have been practically impossible to make the work even approximately complete. The enlightened and liberal management of that library has made the whole world debtors to America, and it should receive even a far more generous support from the Government than it has had.

My Toner Lecture was the first publication in which were gathered together the many surgical results of typhoid fever. Before its publication only single surgical lesions had been considered or sporadic cases had been published. Unfortunately, not having appeared in any of the medical journals, it never became well known to the profession. I have, therefore, with the kind permission of the Smithsonian Institution, republished it in the Appendix.

CHAPTER II.

PATHOLOGY OF THE SURGICAL COMPLICATIONS AND SEQUELS OF TYPHOID FEVER.

THE province of the physician and that of the surgeon are, in general, sufficiently sharply defined and differentiated, yet they have many points of contact. While some diseases belong exclusively to the province of the one and some to that of the other, other diseases may fall with equal propriety under the care of either practitioner. Still another class of cases, however, beginning in the domain of medicine may terminate in that of surgery, and we may lack their complete history from the very fact of this division of their care and interest.

Among the diseases classed as strictly medical, none deserve the appellation more definitely than the continued fevers, and especially typhoid fever. Yet the present work shows that these fevers are not infrequently the cause of the gravest and least expected surgical troubles, mention of which is generally omitted, even in our best text-books on medicine, still more rarely noticed in works on surgery, and, where noticed, it is only with the greatest brevity.

The surgical results of scarlet fever and the other exanthemata have long been recognized and described. But up to 1876, when I published my Toner Lecture, surgical troubles after typhoid

and other continued fevers were deemed rather among the curiosities of literature, than looked upon as somewhat rare, but yet well-ascertained surgical post-febrile lesions, which should be expected from time to time and, therefore, watched for and, if possible, prevented. Since 1880, when Eberth discovered the bacillus of typhoid fever, our views of the pathology not only of the fever itself, but also of its complications and sequels, recent and remote, have undergone an entire revolution and we now understand much that was formerly inexplicable.

I shall only consider the pathology of typhoid sufficiently for us to see its bearing from the surgical side; more than that would be out of place. The pathology of typhus fever is so much less clear and certain, and the number of cases after typhus so much smaller, especially at the present time, that I shall entirely omit its special consideration.

In order to gain a clear idea of the pathology of typhoid and post-typhoid lesions, I will consider:

1. The viability of the typhoid bacilli both in and out of the body; and, therefore, the possibility of their causing late as well as early sequels of the fever.

2. Their wide diffusion in the various organs of the human body, and, therefore, the possibility, if not the probability, that all the various surgical results may be caused by them.

3. Mixed infections of the typhoid bacilli with other bacteria.

4. The pyogenic faculty of the typhoid bacilli.

5. Typhoid infection of different organs without typical typhoid lesions in the intestine.

I. **The Viability of the Typhoid Bacilli** (*a*) **Outside the Human Body.**—In favorable conditions—*e. g.* in a bacteriological laboratory—the typhoid germs may live indefinitely: but in unfavorable conditions, also, their viability is remarkable. Thus Lösener [1] found that the bacillus only disappeared from the body ninety-six days after burial. In feces they may live even for months.[2] Remlinger and Schneider [3] have found them in the feces of five out of ten patients who had never had typhoid fever.

Schiller [4] impregnated threads with the bacillus, and found them still active in periods of from one to two years.

Uffelmann [5] found them still living and active after eighty-two days in dry sterile sand.

(*b*) **Their Viability Inside the Human Body.**[6] —I will not mention instances, of which there are a large number, in which the typhoid bacilli have been found in abscesses, etc., within three or four weeks after the fever, but only those cases in which they have been found months and even years afterward.

By far the longest time after the fever, when the

[1] Arbeit. kaiserlich. Gesundheitsamte, 1895.

[2] Ziegler, Allgem. Pathol., 1895, i, 597.

[3] Ann. de l'Institut Pasteur, 1897, xi, 55.

[4] Arbeit. kaiserlich. Gesundheitsamte, v, 312.

[5] Centralbl. f. Bakteriol., 1894, Nos. 5 and 6.

[6] Schnitzler, Centralbl. f. Bakteriol. und Parasitenk., 1894, 270, in a case of old osteomyelitis, found the staphylococcus pyogenes aureus after thirty-five years, and Krause, Fortschritte d. Med., ii, Nos. 7 and 8, found the same organism after thirty years. Both these, however, may have resulted from a continuous or a late re-infection. I merely mention them as instances of the extreme viability of these organisms also in the human body.

typhoid bacillus has been found, and in pure culture, is that of von Dungern.[1] Fourteen years and a half after the fever the bacillus was found in an abscess about the gall-bladder. A résumé of the case is given in Chapter XVI.

Buschke[2] reports the case of a woman of sixty-six who fell ill of typhoid in October, 1886. Before Christmas, when she left her bed, she complained of swelling, pain, and tenderness in the fifth and sixth ribs and cartilages on the right side. The pain and tenderness subsided, but the swelling did not disappear. In December, 1893, without any evidence of a re-infection, the pain returned, but again subsided only to recur for a third time. On her admission to the hospital February 19, 1894, below and to the inside of the right breast was a swelling the size of a hen's egg, soft and fluctuating in parts. In the axilla was an enlarged gland. On February 26th the swelling was incised, the pus evacuated, and the granulation tissue curetted. A pure culture of the typhoid bacillus was found in the pus. Hence over seven years after the fever the bacilli had retained their vitality and their virulence, as was shown not only by the abscess in the patient, but by experiment. Bouillon cultures of these bacilli were injected into rabbits. These rabbits, it is true, remained well, but from the places where the injections were made new cultures were taken, with every antiseptic precaution, and these injected in other rabbits proved fatal, and from their dead

[1] Münch. med. Wochen., 1897, No. 26, p. 699.

[2] Lebensdauer d. Typhusbacillen in ostitischen Herden, Fortschritte d. Med., 1894, 573.

bodies the typhoid bacillus was obtained, thus completing the circle of proof.

Bruni[1] records the case of a woman of thirty-six who, in 1889, during convalescence, developed an osteoperiostitis of the right femur with tenderness over the left tibia. Six years later an osteomyelitis developed in the latter bone. The tibia was trephined, and from the pus in the medullary cavity typhoid bacilli were obtained and their virulence established by inoculation.

The next longest case on record is that of Sultan.[2] For six years after the fever the patient had had a fistulous opening leading down to the clavicle. Though an infection with the ordinary pyogenic or other extraneous bacteria would seem to be inevitable in a case of open suppuration extending over so long a period, the discharge showed a pure culture of the typhoid bacillus. Chantemesse[3] has described a case with abscesses in the right tibia, left femur, left ulna, and a left finger in which a pure culture of the typhoid bacillus was found after four years.

In addition to these extraordinary cases, the following authors have found the bacilli, usually in pure culture, after long periods. Parsons[4] reports a pure culture in the case of a sinus open and discharging for three months; Péan and Cornil,[5] in a tibial periostitis and abscess after eight months; Chantemesse,[6] in a similar case, after six and a half months, and in

[1] Ann. de l'Institut Pasteur, 1896, x, 220.
[2] Deutsch. med. Wochen., 1894, 675.
[3] Bull. et Mém. Soc. Méd. des Hôp., 1893, x, 779.
[4] Johns Hopkins Hosp. Reports, v, 417.
[5] Bull. Acad. Méd., Paris, 1891, 3d series, xxv, 599.
[6] Bull. et Mém. Soc. Méd. des Hôp., 1893, x, 779.

another after eleven months; Chantemesse and Widal,[1] in an abscess after fifteen months; Orloff,[2] in a case of periostitis after eight months; Klemm,[3] in an osteomyelitis after a year; Chantemesse and Widal,[4] after eighteen months; Werth,[5] in an ovarian cyst after eight months; Berg,[6] in a case of chondritis of the costal cartilages after three years; Lockwood,[7] in a tibial abscess after fifteen months; Hintze,[8] in a "cold" periosteal abscess after ten months; Fraenkel,[9] in a pleural effusion after five months, and in an abdominal abscess[10] after four and a half months.

Of course, in many cases the typhoid bacilli have not been found. Thus, Schede[11] was unable to find them in ten cases of abscess of bone. All were due to the pyogenic bacteria. But such cases, however numerous they might be, could not change the facts above cited in which there is positive and cumulative proof of the long-continued vitality and virulence of the typhoid bacilli in various tissues, but especially in the bones. That they may be, and not only may be but certainly are, responsible for the mischief in the midst of which they were found, even though it be long after the fever, is a most reasonable conclusion. This is rendered still more certain by the experimental proof of the pyogenic power of the typhoid bacilli shortly to be given (p. 40).

[1] Traité de Méd., July, 1891, i. [2] Deutsch. med. Wochen., 1890, p. 1806.
[3] Arch. klin. Chir., 1893, xlvi, 862. [4] Sem. Méd., 1893, 542.
[5] Deutsch. med. Wochen., 1893, No. 21.
[6] Centralbl. f. Chir., 1896, 153.
[7] Lancet, 1895, i, 531. [8] Centralbl. f. Bakteriol., Oct. 10, 1893, 445.
[9] Buschke, *loc. cit.*, 581. [10] Deutsch. med. Wochen., 1894, p. 152.
[11] Münch. med. Wochen., 1888, No. 11.

II. **The Wide Diffusion of the Typhoid Bacilli in Various Organs and Tissues of the Body.**—Scarcely a single tissue or organ of the body escapes invasion by the typhoid bacilli. Not only the normal tissues are invaded, but, as is seen by the preceding section and in many later chapters, they are found in the pus of abscesses in every region of the body and in empyema, in the effusion of teno-synovitis, in the non-purulent fluid of orchitis, etc. Among the various tissues and organs in which the bacilli have been found are the following:

1. **The Blood.**—The bacilli of typhoid are not commonly found in the blood. When present, they are most numerous in the first twelve days of the disease. From then until the end of the third week they diminish rapidly, and during the fifth and sixth weeks are only found exceptionally (Park). It is probable that they reach the blood by the lymphatics, since they are found in abundance in the thoracic duct.[1] That they must be distributed to other parts of the body (*vide infra*) by the blood (though so rarely actually found there) is made probable also by the fact that not rarely they are found in multiple organs of the body which could only be reached through the circulation. This is strikingly shown in the remarkably well-studied cases of Flexner,[2] in which he found them in the mesenteric glands, the spleen, the liver, the bile, the kidneys, the lungs, the marrow of the bone, and in the blood of the heart. In the kidneys there were a large number of abscesses resembling miliary tuber-

[1] Déhu, Le rôle du bacille d'Eberth dans les complications de la fièvre typhoide, Thèse de Paris, 1893. p. 59.

[2] Jour. of Pathol. and Bacteriol., 1894, iii, 202.

cle, which, however, were proved to be real abscesses containing the bacillus of typhoid in pure culture.

Vincent[1] also gives details of six cases in which they were found in the blood, the spleen, the liver, the mesenteric glands, the brain, the spinal cord, the cerebrospinal fluid, the kidneys, and the lungs.

Neuhauss,[2] Rütimeyer,[3] Achalme,[4] Kühnan,[5] Anderson,[6] Guarnieri,[7] and Block[8] have also found them in the blood, and in some instances proved their activity by injection into animals.

Kanthack and Tickell[9] and Ohlmacher[10] have found them in the blood in mixed infection with the streptococcus.

That they must exist in the blood and be diffused by it is also proved by several cases in which the fever, having caused the patient to abort, the bacillus of Eberth has been found in the placenta and the fetus (p. 32). In these cases no other route for the infection is possible. Chantemesse and Widal[11] also found the bacilli in large quantities in the fetus and the amniotic fluid of the cobaye.

2. **In the endocardium and endocardial vegetations.**

3. **In the walls of the arteries and the veins,** producing arteritis and peri-arteritis, phlebitis and periphlebitis.

[1] Ann. de l'Institut Pasteur, 1893, vii, 141.
[2] Berlin. klin. Wochen., 1886, Nos. 6 and 24.
[3] Centralbl. f. klin. Med., 1887, No. 9.
[4] Comptes Rendus Soc. de Biol., June 21, 1890.
[5] Berlin. klin. Wochen., June 23, 1896.
[6] Med. News, Aug. 8, 1896, 155. [7] Revist. Gen. Clin. Med., 1892, 234.
[8] Johns Hopkins Hosp. Rep., June, 1897.
[9] Edinb. Med. Jour., July, 1897, 22.
[10] Cleveland Med. Gaz., May, 1897.
[11] Gaz. Hebdom., March 4, 1887, 146.

4. **In thrombi** in the heart arteries and veins.

These three are considered more fully in Chapter III, on Gangrene, since they bear especially upon that process.

5. **In the muscles** they have been found by Gasser,[1] Raymond,[2] Rosin and Hirschel,[3] Jahradnicky.[4]

6. **In the Connective Tissues.**—There are only three cases in our table in which a bacteriological examination has been made. In Vincent's case[5] the typhoid bacilli were found in the cardiac vegetations and the blood in the heart, in the spleen, kidney, brain, bone-marrow, etc., but no observations were made as to the muscular hematoma. In Lockwood's,[6] the pus of an abscess over the tibia contained only the bacilli of Eberth fifteen months after the fever.

Raymond,[7] in 1890, after a careful examination by Strauss, reported that the bacillus of Eberth was found in pure culture. Among other such muscular cases are those of Swiczinki[8] and Tinctine.[9]

7. **Skin.**—Most of the infections of the skin are probably pyogenic, but Rheiner[10] has found the typhoid bacillus in the skin in cases of post-typhoid erysipelas. See also Valentini[11] and Raymond[12] for other facts. Sittmann[13] records a curious case of a suppurating lipoma of the skin of the knee in which the typhoid bacillus was found in pure culture.

[1] Pein, L'Action pyogénique du bacille typhique, p. 88. [2] *Ibid.*, p. 58.
[3] Deutsch. med. Wochen., 1892, No. 22. [4] Centralbl. f. Chir., 1896, 336.
[5] Merc. Méd., Feb. 17, 1892, p. 73. [6] Lancet, 1895, i, 531.
[7] Pein, Thèse de Paris, 1891, p. 58. [8] Chronica Lekarska, 1894, No. 8.
[9] Arch. de Méd. Exp. et d'Anat. Pathol., 1894, vi, 1.
[10] Arch. f. Path. Anat., 1885, c, 185.
[11] Berlin. klin. Wochen., 1889, No. 17.
[12] Gaz. Méd. de Paris, 1891, No. 9.
[13] Deutsch. Archiv f. klin. Med., liii, 1894, 352.

8. **Synovial sheaths** of tendons at ankle. Grancher[1] reports the only case I have found.

9. **Joints.**—In Chapter IV I have given the results of all the bacteriological examinations I have myself made or found recorded. They are all negative so far as finding the typhoid bacillus, except that they were once recorded as present soon after experimental inoculation into the joints.

10. **Bones.**—Very many observers, as indicated in Chapter V, have found the bacilli in the marrow of the bones, where they have remained for a greater or less time without producing disease. Usually these cases have been observed either at post-mortem examinations or in experimental researches. In other cases the bacilli have been found in periostitis, abscess, osteomyelitis, etc. I do not pretend to give a complete list, but among them may be named:

Ebermeier, Deutsch. Arch. f. klin. Med., 1889, xliv, 140.

Flexner, Jour. of Pathol. and Bacteriol., Nov., 1894, iii, 202.

Orloff, Wratsch, 1889, No. 49, and 1890, Nos. 4, 5, and 6.

Valentini, Berlin. klin. Wochen., 1889, No. 17, 368.

Achalme, Comptes Rendus Soc. de Biol., June 21, 1890, 387.

Chantemesse and Widal, Arch. de Physiol., 1887.

Chantemesse, Bull. et Mém. Soc. Méd. des Hôp., 1890, vii, 655; 1893, x, 779.

Bauer, Centralbl. f. Chir., 1895, 788.

Colzi, Lo Sperimentale, lxv, 1890, 623.

Melchior, Jahresbericht Fortschritte Pathol. Mikroörgan., Band viii.

[1] Bull. Méd., 1892, vi, 1271.

Dupraz, Arch. Méd. et Anat. Pathol., Jan. 1, 1892, 76.
Buschke, Fortschritte d. Med., 1894, 573.
Sultan, Deutsch. med. Wochen., 1894, 675.
Parsons, Johns Hopkins Hospital Rep., v, 417.
Gilbert and Girode, Comptes Rendus Soc. de Biol., July 11, 1890, and May 2, 1891.
Klemm, Arch. f. klin. Chir., 1893, xlvi, 862; xlviii, 792.
Péan and Cornil, Bull. Acad. de Méd., 1891, 3d series, xxv, 599.
Quincke, Berlin. klin. Wochen., 1894, No. 15.
Hintze, Centralbl. f. Bakteriol., 1893, 445.
Helferich, Berlin. klin. Wochen., 1890, 979.
Vincent, Merc. Méd., Feb. 17, 1892, 73.
Berg, Centralbl. f. Chir., 1896, 153.
Rosin and Hirschel, Deutsch. med. Wochen., 1892, 493.
Welch, N. Y. Med. Record, 1893, i, 631.
Barbacci, Lo Sperimentale, 1891, 356.
Mouisset, Lyon Méd., 1891, 326.

11. **In the brain and spinal cord** (meningitis, abscess, etc.):

Ohlmacher, Jour. of Amer. Med. Assoc., Aug. 28, 1897, xxix, 419.
Kamen, Internat. klin. Rundschau, 1890, Nos. 3 and 4.
Neumann and Schaeffer, Virchow's Archiv, 1887, cix, 480.
Honl, Centralbl. f. Bakteriol. und Parasitenk., xiv, 767.
Adenot, Lyon Méd., 1889, Nos. 34 and 36, and Arch. de Méd. Exp. et d'Anat. Pathol., 1889, No. 5.
Hintze, Centralbl. f. Bakteriol., Oct. 10, 1893, 445.
Mensi and Carbone, Riforma Med., 1893, No. 2.
Silva, Riforma Med., 1891.

Vincent, Merc. Méd., Feb. 17, 1892, p. 73.
Tinctine, Arch. de Méd. Exp. et d'Anat. Pathol., 1894, vi, 1.
Fernet, Bull. Soc. des Hôp., 1891, No. 23, 361.
Daddi, Lo Sperimentale, 1894, No. 17, 325.
Balp, Revist. Gen. Ital. Clin. Med., 1890, No. 17, 406.
Chantemesse and Widal, Gaz. Hebdom., 1887, 146.
Curschman, Verhandl. Kongress f. inner. Medizin, 1886, 469.
Kühnan, Berlin. klin. Wochen., 1896, June 23d.

12. **In the thyroid gland :**

Colzi, Revist. Gen. Clin. Med., 1891, No. 10, and Lo Sperimentale, 1891, No. 2.
Honl, Centralbl. f. Bakteriol. und Parasitenk., xiv, 767.
Jeansalme, Arch. Gen., 1893.
Spirig, Correspondenzbl. schweiz. Aerzte, Feb. 15, 1891.
Dupraz, Arch. de Méd. Exp. et d'Anat. Pathol., Jan. 1, 1892, p. 76.
Kummer and Tavel, Rev. de Chir., June, 1891, and Aetiol. d. Strumitis, Basel, 1892.

13. **In the orbit :**

Panas, Cinquième Congrès de Chir., March 30, 1891.

14. **In the parotid gland :**

Lehmann, Centralbl. f. klin. Med., Aug., 1891, 649.
Janowski, Centralbl. f. Bakteriol. und Parasitenk., 1895, xvii, 785.

15. **In otitis media :**

Destrée, Jour. Méd. Brux., xcii, Aug. 15th.
Vincent, Ann. de l'Institut Pasteur, Paris, 1893.

16. **In the heart-muscle:**

Chantemesse and Widal, Gaz. Hebdom., 1887, 146.

17. **In the lungs:**

Flexner, Jour. Path. and Bacteriol., Nov., 1894, iii, 206.

18. **In the pleura** (empyema, etc.):

Charrin, Soc. Méd. des Hôp., March 17, 1891. Infection of the pleuritic fluid with the typhoid bacillus, but no intestinal lesions of typhoid.

Fernet, Bull. Soc. Méd. Hôp., 1891, May 21, 236.

Loriga and Pensuti, Riforma Med., 1890, 1232.

Valentini, Berlin. klin. Wochen., 1889, 368.

Spirig, Mittheil. aus klin. und med. Instit. d. Schweiz, Basel, 1893–94, 771.

Kelsch, Merc. Méd., March 2, 1892, p. 97.

Weintraud, Berlin. klin. Wochen., 1893, xxx, 345.

Rendu and de Gennes, La France Méd., 1885, ii, 1821,

Belfanti, Revist. Gen. Ital. de Clin. Med., 1890, No. 20.

Foa and Bordoni-Uffreduzzi, Riforma Med., 1887, No. 1.

Burci, Arch. Ital. clin. Med., 1893, xxxii, 1.

Arustamoff, Centralbl. f. Bakteriol. und Parasitenk., vi, 1889.

19. **In the peritoneum:**

Fraenkel, Sem. Méd., April 27, 1887.

Lehmann, Centralbl. f. klin. Med., Aug., 1891, 649.

Raymond, Sem. Méd., 1891.

Fraenkel, Deutsch. med. Wochen., 1894, 152.

20. **In the liver:**

Guarnieri, Revist. Gen. Clin. Med., 1892, 234.

Chantemesse and Widal, Gaz. Hebdom., 1887, 146.

Chantemesse, Bull. et Mém. Soc. Méd. des Hôp., 1893, x, 779.

Cygnäus, Ziegler's Beiträge, etc., 1890, vii.
Flexner, Jour. Path. and Bacteriol., Nov., 1894, iii, 202.
Lannois, Rev. de Méd., 1895, No. 11, 909.
Vincent, Merc. Méd., Feb. 17, 1892, 73.
Rosin and Hirschel, Deutsch. med. Wochen., 1892, 493.
Gaffky, Mitth. kaiser. Gesundheitsamte, ii, 1884.
Fraenkel and Simmonds, Die ætiolog. Bedeut. d. Typhusbacillen, Leipzig, 1886.

21. **In the gall-bladder and bile:**

Von Dungern, Münch. med. Wochen., 1897, No. 26.
Osler, see Chapter XVI.
Chantemesse, Traité de Méd., i, 764.
Mason, Boston Med. and Surg. Jour., May 13, 1897, and Trans. Assoc. Amer. Phys., 1897.
Gilbert and Girode, Sem. Méd., 1890, No. 58, and Mém. Soc. Biol., 1893, 956.
Chiari, Prag. med. Wochen., 1893, No. 22, 261.
Dupré, Les Infections Biliaires, Thèse de Paris, 1891.
Fournier, Thèse de Paris, 1896.
Salzmann, Württemb. med. Correspondenzbl., 1870, p. 84.
Flexner, Jour. Pathol. and Bacteriol., Nov., 1894, iii, 202.
Welch, Johns Hopkins Hosp. Bull., Aug., 1891, No. 15, 121.
Blackstein, Johns Hopkins Hosp. Bull., No. 14, p. 96.
Alexieef, Amer. Jour. Med. Sci., Oct., 1897, 466.

22. **In the spleen:**

Flexner, Jour. Pathol. and Bacteriol., Nov., 1894, iii, 202.
Ohlmacher, Jour. Amer. Med. Assoc., August 28, 1897, xxix, 419.

Kanthack and Tickell, Edinb. Med. Jour., July, 1897, 22.
Vincent, Merc. Méd., Feb. 17, 1892, 73.
Roux and Vinay, Lyon Méd., June 10, 1888.
Neuhauss, Berl. klin. Wochen., 1886.
Chantemesse and Widal, Gaz. Hebdom., 1887, 146.
Ebermeier, Deutsch. Arch. f. klin. Med., 1889, xliv, 140.
Laveran, Bull. Soc. Méd. des Hôp., 3d series, vol. viii, p. 90.
Haushalter, Merc. Méd., Sept. 20, 1893. 453.
Kühnan, Berl. klin. Wochen., June 23, 1896.
Guarnieri, Revist. Gen. Clin. Med., 1892, 234.

23. **In the mesenteric glands:**

Flexner, Jour. Pathol. and Bacteriol., Nov., 1894, iii, 202.
Ohlmacher, Jour. Amer. Med. Assoc., August 28, 1897, xxix, 419.
Lehmann, Centralbl. f. klin. Med., August, 1891, 649.
Fraenkel, Sem. Méd., 1887, 173.
Vincent, Merc. Méd., Feb. 17, 1892, 73.
Gilbert and Girode, Comptes Rendus Soc. Biol., May 2, 1891.
Kühnan, Berl. klin. Wochen., June 23, 1896.

24. **In the kidney:**

Flexner, Jour. Pathol. and Bacteriol., Nov., 1894, iii, 202.
Faulhaber, Beitr. path. Anat. und allg. Path., x.
Koujojeff, Centralbl. f. Bacteriol. und Parasitenk., 1889.
Vincent, Merc. Méd., Feb. 17, 1892, 73.
Von Wünscheim, Prag. med. Wochen., xliv, 1894.
Seitz, Centralbl. f. Bakteriol., ii, 1887.
Neumann, Berlin. klin. Wochen., 1890.

25. **In the urine:**

Bouchard, Rev. de Méd., 1881, 671.

Kanthack and Tickell, Edinb. Med. Jour., July, 1897, 22.

Wissokowitsch, Zeitschr. f. Hyg., vol. i, p. 1.

Seitz, Bakteriol. Studien z. Typhusaetiologie, 1886.

Hueppe, Fortschritte d. Med., 1886, 447.

Koujojeff, Centralbl. f. Bakteriol. u. Parasitenk., 1889, vi, 24.

Neumann, Berlin. klin. Wochen., 1888, No. 7, 1890, No. 6.

Faulhaber, Ziegler's Beitr. path. Anat. u. allg. Pathol., x, 1891.

Karlinski, Prag. med. Wochen., 1890, xv, 437.

26. **In the ovary:**

Werth, Deutsch. med. Wochen., 1893, No. 21.

Sadeck, Münch. med. Wochen., 1896, No. 21.

27. **In the testicle and epididymis:**

Girode, Arch. Gén., 1892, i, clxix, 43.

Tavel, Correspondenzbl. schweiz. Aerzte, 1887, 590.

Gasser, Arch. de Méd. et de Pharm. Milit., 1895, No. 3, 228.

Ménétrier in Pein, Thèse de Paris, 1890, p. 18.

Belfanti, Revist. Gen. Ital. Clin. Med., 1890, No. 20.

28. **In the placenta and fetus:**

Widal and Chantemesse, Gaz. Hebdom., March 4, 1887, 146.

Hildebrand, Fortschritte d. Med., 1889, vii, 889.

Eberth, Fortschritte d. Med., 1889, vii, No. 5.

Ernst, Beitr. path. Anat. u. allg. Path., 1890, viii, p. 1.

Etienne, Gaz. Hebdom., 1896, No. 16.

Janizewski, Münch. med. Wochen., 1893.

Frascanni, Jahresb. Fortsch. Path. Microörganism., 1892.
Neuhauss, Berl. klin. Wochen., 1886.
Reher, Arch. Exp. Pathol. et Pharmacol., xx, 420.
Giglio, Centralbl. f. Gynekol., 1890, No. 46.[1]

Griffith[2] reports an interesting case of a child born at full term while the mother was still ill of typhoid fever. The attack was light, the rose spots appeared on November 13th, and the child was born on the 18th. When the child was seven weeks old it responded to Widal's test. He concludes that the child had had an attack of the fever *in utero*.

A still more remarkable case is reported by Etienne.[3] He examined a five months fetus born of a mother aged eighteen, on the twenty-ninth day of typhoid. "The child's spleen and intestines, as well as other organs, showed no evidence of the disease, and the placenta was healthy. Blood from the right side of the heart and from the spleen, liver, and placenta was carefully examined and cultures made. The typhoid bacillus was found in abundance. The fetus had really died of typical acute blood-poisoning from a large dose of the bacillus before the occurrence of any local change."

III. **Mixed Infection of the Typhoid Bacillus and the Pyogenic and Other Bacteria.**—Very rarely specific infections, such as those of tetanus, erysipelas, anthrax, and malignant edema, occur in

[1] In this bibliography I have been materially assisted by Flexner's paper, Ziegler's allgemein. Pathol., Dmochowski and Janowski's, and Rosin and Hirschel's papers.

[2] Med. News, May 15, 1897.

[3] Brit. Med. Jour., Epitome 1896, ii, 35, from the Gaz. Hebdom., 1896, No. 16.

cases of typhoid fever. These mixed infections are considered in Chapter XIX. In this section I shall only review the relation of the typhoid and the pyogenic bacteria. The subject can not be sharply separated from that of the next section—cases of suppuration caused by the typhoid bacillus in pure culture—but will well lead up to it.

Many authors, especially those who made the earlier bacteriological examinations, disbelieved absolutely—and very naturally—in the pyogenic properties of the typhoid bacillus. They ascribed all suppurative lesions during or following the fever to an infection by the pyogenic bacteria alone or to a superadded or secondary infection by the pyogenic bacteria in addition to that by the typhoid bacillus.

Many surgical affections during and after typhoid are undoubtedly almost constantly purely pyogenic. For example, otitis media almost always results, so far as we know, from pyogenic bacteria; parotitis in the great majority, and the same is true of abscesses, etc. In many cases even of disease of the bones this is equally the fact—*e. g.*, Schede[1] in ten cases of abscess of bones found only the pyogenic bacteria, and Dunin[2] found the same bacteria in eight cases of lesions of bones.

Nay, more than this, the pyogenic infection may, though very rarely, result in distinct pyemia or septicemia in which the typhoid bacilli play no part whatever. Thus one case reported by Wagner[3] com-

[1] Münch. med. Wochen., 1888, No. 11.
[2] Deutsch. Arch. f. klin. Med., 1886, xxxix, 369.
[3] British Med. Jour., 1891, i, 18.

menced with a crural phlebitis relatively early in the disease (the ninth day), death occurring from pyemia on the eighteenth day. In another, reported by Spillman,[1] gangrene of the lips occurred, probably due to carious teeth, and the patient died from staphylococcus septicemia, the aureus being found in the spleen, kidneys, and liver. It had probably obtained entrance from the mouth and set up a secondary systemic infection during convalescence as the earlier infection was disappearing. Rendu[2] reports another case with purulent arthritis of the right hip-joint, abscesses of the right thigh, and osteomyelitis, purulent pleuro-pneumonia, and nephritis. Julliard[3] reports a case of septicemia following a typhoid hemorrhage in the neck.

That mixed infection should occur in typhoid fever is not at all remarkable, since the intestinal ulcers and, in a large minority of the cases also, the ulceration of the mucous membrane of the mouth, and the bedsores which are so frequent in typhoid, afford favorable ports of entry.[4]

Mixed infections, by co-existing pyogenic and typhoid bacteria, as would be expected, are not uncommonly met with. Thus Lehmann[5] reports a fatal case of encapsulated peritoneal abscess and suppurative parotitis, and in the parotid was found a mixed culture of the Eberth bacillus and the staphylococcus.

[1] Merc. Méd., 1895, No. 13, 145.

[2] Witzel, Gelenk-u. Knochenkrankh.

[3] Guyot, Étude sur l'Hématocèle périutérine, etc., dans la Fièvre Typhoide, Thèse de Paris, 1879.

[4] See Introduction for a fuller discussion of these points.

[5] Centralbl. f. klin. Med., 1891, 649.

Spirig [1] records a case of suppuration in the thyroid with a mixed culture of the typhoid bacillus and the staphylococcus albus, and Parsons [2] records a case of osteomyelitis and necrosis of the left radius in which the citreus and the typhoid bacillus co-existed, while the aureus alone caused a suppurative periostitis of the right tibia. Rendu [3] found, in the pus of a typhoid empyema, both the pyogenic and the typhoid bacteria.

In a few cases a mixed infection occurs in which the typhoid bacillus exists in one place and a pyogenic organism in another. Thus Laveran [4] records a case of abscesses on the dorsum of the right hand, right arm, and left forearm in which the staphylococcus only was found in these abscesses with great numbers of the typhoid bacilli in the spleen. Flexner [5] reports a case of very wide diffusion of the typhoid bacilli in other organs, but in the parotid gland only the streptococcus was found.

It has been suggested that those cases in which suppuration occurred and the typhoid bacillus alone was found, were due to a primary mixed infection from the pyogenic bacteria and the typhoid bacilli, but that the former had perished after producing the suppuration, leaving the typhoid bacillus alone in the pus.

But in some cases, as Kamen [6] has pointed out, the interval that elapsed was too short (less than

[1] Correspondenzbl. schweiz. Aerzte, Feb. 15, 1891.
[2] Johns Hopkins Hosp. Reports, vol. v.
[3] La France Méd., 1885, ii, 1821.
[4] Bull. Soc. Méd. des Hôp., 3d series, vol. viii, p. 90.
[5] Jour. Pathol. and Bacteriol., 1894, iii, 202.
[6] Internat. klin. Rundschau, 1890, Nos. 3 and 4.

two weeks) for us to suppose that the pyogenic bacteria could have died out; and the result of experiments upon animals and the now very numerous cases in which the typhoid bacillus alone has been found in man preclude the probability of this assumption.

The results noted in the various chapters following, show that it is specifically stated that "pure cultures" of the typhoid bacilli were found in 63 out of 82 cases of suppuration of various kinds. In making the résumés, undoubtedly the word "pure" was sometimes accidentally omitted by us, so that the truth is probably under, rather than over, stated. The extreme disproportion in the figures as they stand, however, is a most convincing proof of the unquestionable pyogenic property of the typhoid bacillus.

Experiments upon animals have pointed positively also in the same direction.

Both of these points are more fully considered in the next section of this chapter. This, however, is a favorable opportunity to consider the results of laboratory researches to determine the behavior of the pyogenic bacteria in mixed cultures, especially the staphylococcus and the streptococcus.

Vincent,[1] in mixed cultures of the bacillus of Eberth and the staphylococcus or the streptococcus, has observed the greatest difference in the influence of these two pyogenic organisms on the bacillus of typhoid. The staphylococcus is remarkably inimical to the growth of the bacillus of typhoid, so that the latter will disappear from a mixed culture, while, on

[1] Ann. de l'Institut Pasteur, 1893, vii, 141.

the contrary, when mixed with the streptococcus, the typhoid bacillus grows vigorously. In his experimental researches he found that doses of the streptococcus or of the bacillus of typhoid, neither of which was fatal if injected singly, if injected together would produce the most violent reaction and death. In 41 cases of typhoid in which various suppurative processes occurred, in 32 the complication was due to the aureus or albus. All of these recovered in spite of extensive suppuration and multiple periostitis. On the other hand, of eight cases in which the streptococcus either alone or associated with the bacillus of Eberth was found, five died, showing a striking difference in the fatality especially of the mixed infection by these two pyogenic organisms.

Valliard[1] says that if two animals are infected, the first with a feeble dose of the streptococcus and the second with a similar dose of the typhoid bacillus, both will be ill for a few days and then recover, but that if a third animal is infected with the same dose of the two bacilli together death follows with stupor, diarrhea, and fever. Dr. Kyle informs me that he has observed the same fatality after mixed infection with the streptococcus and other bacteria.

Orloff[2] also has shown that six weeks after an injection of mixed cultures of the typhoid bacillus with the albus and the aureus, the latter was not destroyed but that in one third of the cases the albus had disappeared.

[1] Quoted by Gamaleia, Bul. Med., 1890, vi, 1273.
[2] Centralbl. f. Chir., 1890, p. 48.

The acknowledged difficulty of distinguishing between the colon bacillus and that of typhoid is also alleged as a reason for doubting the pyogenic faculty of the latter and to ascribe the suppuration to the colon bacillus. But first the number of cases of suppuration which the typhoid bacillus has been found in pure culture (p. 37) is such as to make it very improbable that they could *all* be due to the colon bacillus in so distant and so many organs and tissues; and, secondly, the very exact and careful differentiation of the two bacilli by their various stains and different reactions which, in view of this very objection, have been made by Flexner, Dmochowski and Janowski, Haushalter, Quervain, Bruni, Ohlmacher, and others, would overthrow the objection.

The following remarks by Flexner show that this differentiation is now not only possible but certain:

"The weight of opinion among bacteriologists at this time is that the colon group of organisms can be sharply differentiated from the typhoid bacillus. However, it no longer suffices for distinguishing them to observe the colony growth upon agar-agar, gelatin plates, or the growth upon potato; neither does the possession of mobility and stainable flagella serve to distinguish the typhoid from the colon bacillus. On the other hand, the power of acid and indol production, the property of setting up fermentative changes in sugar, with the liberation of gas and the coagulating influence upon milk, possessed by the colon group of organisms, separate it from the typhoid organism, which produces alkali, does not yield indol in cultures, is not capable of ferment-

ing sugar, and has no coagulating effect upon milk."[1]

In at least once case of superficial necrosis of the femur, Klemm[2] has found both the colon and the typhoid bacilli co-existing. While, therefore, it is possible that the pyogenic bacteria may have been the cause of the suppuration in many of the cases and may have died out in others, it is undoubtedly true that the typhoid bacilli have very frequently a pyogenic faculty.

Why in some conditions the typhoid bacillus should be pyogenic and in others not, we can only at present speculate. No reason can be alleged. But we are precisely in the same position as to the pyogenic function of other bacilli,—for example, the colon bacillus, which we certainly know to be a possible if not indeed a frequent cause of suppuration in certain conditions,—*e. g.*, appendicitis,—whereas ordinarily it is an entirely harmless intestinal organism.

IV. **The Pyogenic Faculty of the Typhoid Bacilli.**—The proof of this lies first in observed cases in man in which the typhoid bacilli have been found in pure culture, and, secondly, in experiments upon animals. The first to observe this pyogenic property of the typhoid bacilli were Rendu,[3] Fraenkel,[4] and Tavel.[5]

[1] Clement, Jour. Pathology and Bacteriology, 1893-94, vol. ii, p. 20. On the same page, in a foot note, he mentions the various recent methods of distinguishing between the two bacilli.

[2] Arch. f. klin. Chir., xlviii, 1894. [3] La France Méd., 1885, ii.

[4] Siebente Kongress Inn. Med., 1888.

[5] Correspondenz-Bl. schweiz. Aerzte, 1888.

(*a*) **In Man.**—The objections have been considered in the preceding section.

As already stated, there are at least 63 cases in which the typhoid bacillus alone existed in various suppurative disorders, and 19 more in which the typhoid bacillus was found, but no mention is made—at least in the table—as to whether other organisms were sought and found. It is, however, reasonable to conclude that any careful bacteriologist would not limit his search to the typhoid bacillus alone, and that had other organisms been found, it would have been so stated. Nearly all of the 82 cases taken together, therefore, were probably cases in which the typhoid bacillus existed in pure culture in suppurative lesions.

That they were also the direct cause of the suppuration both the number of the cases and the other confirmatory evidence seem to prove. Certainly the burden of proof lies upon the opponents of this reasonable view.

Not only the number of these cases, but their wide distribution, is worthy of note. The typhoid bacilli have been found in pure culture in suppurative lesions in the ribs, clavicle, radius, ulna, femur, tibia, and the bones of the fingers and the toes; in the blood, heart-muscle, the endocardium, and the arteries and the veins of the legs; in the muscles of the neck, shoulder, abdominal wall, and legs; in the peritoneal cavity, the spleen, the liver, the bile, the kidney, the urine, and the mesenteric glands; in the brain; in the parotid gland; in the thyroid gland; in the lungs and the pleura; in the ovary; in the testicle.

(*b*) **In Animals.**—There is now a large number of experiments in which the injection of the typhoid bacillus in pure culture, obtained from human subjects, has produced suppuration in animals. No other known cause existed for the suppuration, and the inference that the typhoid bacillus was the cause is unavoidable.

The present work is scarcely the place in which to discuss this subject in detail, but those interested in the subject may consult the following authors who have furnished such proof of the pyogenic faculty of the typhoid bacilli. The somewhat extensive list—and it could have been much enlarged—shows that this property of the bacillus of Eberth has received experimental support from very many careful observers:

Sirotinin, Zeitschr. f. Hyg., i, 1886.

Beumer and Peiper, Zeitschr. f. Hyg., i, 1886, and ii, 1887.

Colzi, Lo Sperimentale, 1890, lxv, 623.

Buschke, Fortschritte d. Med., 1894, xii, 613.

Gaffky, Mitth. kaiserl. Gesundheitsamte, Berlin, ii, 1884.

Fraenkel and Simmonds, Aetiolog. Bedeut. d. Typhusbacillen, Leipzig, 1886.

Seitz, Centralbl. f. Bakteriol., ii, 1887.

Melchoir, Dehu, *loc. cit.*, p. 187.

Achalme, Soc. Biol., June 11, 1890.

Orloff, Pein, *loc. cit.*, p. 70.

Gilbert and Girode, Bull. Soc. Biol., July 11, 1890, and May 2, 1891.

Michon, Des Supp. dans la Fièvre Typhoide, Thèse de Paris, 1890.

Gasser, Pein, *loc. cit.*, p. 88.

Dmochowski and Janowski, Ziegler's Beiträge, xvii, 1895.

Typhoid Infection of Different Organs Without Typical Typhoid Lesions in the Intestine.—A number of cases of typhoid infection in various parts of the body,—very largely surgical,—without any typhoid lesions in the intestine, have been reported. Those which I have met with are as follows:

1. **In the Spleen.**—Du Cazal[1] reports the case of a man, age twenty-one, who had pneumonia and pleurisy, enlarged spleen with great tympany, bleeding from the nose, and headache, but no typhoid spots. He was admitted to the hospital March 2, 1893, and died on the 13th, about the twenty-third day of the disease. Typhoid bacilli in pure culture were found in the spleen, and their identity was confirmed by experiments upon animals. The intestines were entirely free from any typhoid lesion. He refers also to cases by Vincent, Valliard, Chantemesse, Rendu, Fernet, and Kelsch.

2. **In the Spleen, Blood, and Urine.**—Cheadle[2] reports a very carefully examined case of typhoid fever without ulceration of the intestine in which both the "Widal serum-test during life and the bacteriological examination made after the death of the patient rendered the diagnosis absolutely certain." A boy, aged three, was admitted to the hospital November 8, 1896, with a history of diarrhea, vomiting, and debility. A brother and a sister of the patient had been in the same hospital suffering from typhoid fever. Rose-colored spots appeared, the abdomen was swollen and tympanitic, and the

[1] Bull. et Mém. Soc. Méd. des Hôp., 1893, p. 243.
[2] Lancet, 1897, ii, 254.

liver was enlarged but the spleen was not. Ten days after his admission, and probably about the twentieth day of the disease, epistaxis and vomiting occurred. By the twenty-sixth day he was comatose, and died December 1st, about the thirty-third day of the disease.

"On the twenty-first day of the disease, November 19th, on applying the serum-test Mr. MacCallan found that there were loss of motility of most of the bacilli and marked clump formation. The typhoid bacillus was separated from the urine on the twenty-sixth day of the disease by Parietti's method. On the thirty-second day he suspended in five c.c. of water part of a two-day-old stock culture of the bacillus, and to this were added two drops of blood from the patient. The fluid, previously cloudy, became clear after some hours, the bacilli having formed clumps and settled to the bottom of the tube as a flocculent, uneven mass.

"At the post-mortem examination no ulceration of the intestine was visible, and Peyer's patches and the colon appeared to be normal. * * * * The spleen was normal in size and appearance, was opened aseptically, and cultures on agar, gelatin, and potato were made. The culture-tubes after incubation showed typical colonies of the typhoid bacillus. Mr. MacCallan examined microscopically for typhoid bacilli 30 or 40 sections of the spleen, but was unable to recognize any. This was not surprising, as Gaffky says that it is sometimes necessary to examine 200 stained microscopical sections of the spleen in case of typhoid fever before groups of the bacilli can be recognized. The mes-

enteric glands and liver were enlarged. Before the heart was opened, blood was collected from the right auricle in Pasteur pipettes, and the serum-test was carried out with a culture of bacilli which had been separated from the patient's urine. Loss of motility in most of the bacilli and clump formation were observed."

3. **In the Spleen.**—Hodenpyl, in a paper soon to be published in the British Medical Journal,[1] reports the case of a man, age thirty-one, who had all the ordinary symptoms of a "typical typhoid fever, including the rash, of a severe type." He died on the seventeenth day of the disease. The necropsy showed "no enlargement of the mesenteric lymph-nodes. * * * * The mucous membrane of the small intestine was pale. There was neither swelling nor ulceration of the lymphatic structures. Careful examination of Peyer's patches by transmitted light failed to reveal the slightest lesion. The appendix was normal. The colon was dilated and thickly studded with ulcers." The bacteriological examination was made by Dr. E. H. Wilson, bacteriologist to the Brooklyn Board of Health, and later by Drs. Hiss and Dyar, assistants in bacteriology in the laboratory of the College of Physicians and Surgeons, who, working independently of each other, identified the cultures from the spleen as those of the bacillus of typhoid.

4. **In the Spleen.**—Beatty [2] reports the case of a man, age thirty-four, who died on the sixth day of illness. Death was preceded by jaundice, hematuria,

[1] Brit. Med. Jour., 1897, ii, or 1898, i.

[2] Brit. Med. Jour., 1897, i, 148.

and coma. Post-mortem, the spleen and mesenteric glands were enlarged, but the intestines were healthy. From the spleen typhoid bacilli were grown.

He also reports a second case, supposed to be similar to this, but in which no bacteriological examination was made.

5. **In the Spleen.**—Banti[1] reports the case of a woman, age thirty-one, who died on the twenty-sixth day of a typical and apparently uncomplicated attack of typhoid fever. The autopsy showed no lesion of the intestine, but the stained section of the spleen demonstrated bacteria, which Banti regarded as typhoid bacilli.

6. **In the Spleen.**—Karlinski[2] reports three cases, occurring in a wide-spread epidemic of typhoid fever, which presented, during life, the symptoms of this disease. They were affected in much the same way as other fatal cases, which last showed at autopsy the characteristic lesions of the small intestine in typhoid fever. The autopsies of these three fatal cases failed to show any involvement of the lymphatic structures, nor was there swelling or ulceration of either large or small intestines. The mesenteric lymph-nodes and spleen were swollen in each case. In one case the spleen was five times its normal size. Cultivations from the spleen revealed the presence of the typhoid bacillus in each case.

7. **In the Brain.**—Balp[3] reports a case in which the patient entered the hospital, having had a fall which caused unconsciousness and fracture of the humerus.

[1] Riforma Med., 1887, 1448.
[2] Wien. med. Wochen., 1891, 470.
[3] Revist. Gen. Ital. Clin. Med., 1890, 406.

Suppurative meningitis, both spinal and cerebral, was found at the post-mortem. There was a tear of the nasal mucous membrane communicating with the meninges. In the pus from the meninges was found the bacillus of Eberth.

8. **In the Brain.**—Neumann and Schaeffer[1] report the case of a woman who died in the hospital unconscious and without history. No lesions of typhoid were found in the intestine, but from the meninges were obtained pure cultures of the typhoid bacillus.

9. **In the Brain.**—Adenot[2] reports a case in which there was absolutely no lesion found in the kidneys, spleen, mesenteric glands, or intestines, and yet the patient died on the tenth day, five days after the onset of meningitis, and in the fluid infiltrating the brain—for there was no pus—the bacillus of Eberth was found in pure culture.

10. **In Pleuritic Pus.**—Charrin[3] reports the case of a man who died of a purulent pleurisy. The bacteriological examination of the fluid showed the presence of the typhoid bacillus, but none of the intestinal lesions of typhoid were found.

11. **In a Suppurative Goiter.**—Kummer and Tavel[4] report a case in which there was suppuration in an old goiter, due to the typhoid bacillus. The thyroid disease began on the second day. No typhoid bacilli were found in the stools, and no other evidences of typhoid infection were present. The bacteriological examination by Tavel was made, it is true, by older methods, but was most careful and

[1] Virchow's Arch., 1887, cix, 477.
[2] Lyon Méd., 1889, Nos. 34 and 36.
[3] Soc. Méd. des Hôp., March 17, 1891. [4] Rev. de Chir., 1891, 507.

exhaustive. The patient made an excellent recovery.

12. **In the Gall-bladder.**—Osler[1] records a case of acute cholecystitis under the care of Halsted and Cushing. A woman, age twenty-six, who had never had any illness resembling typhoid fever, on March 1, 1897, after an enormous meal, was seized with vomiting, fever, and pain in the region of the gall-bladder, with indistinct tumefaction. On March 16th Halsted operated, finding a large gall-bladder distended with a brownish-tinted material and 15 large stones. She recovered. Cushing made cultures showing the presence of the typhoid bacillus, and the patient's blood gave the characteristic indol reaction to this and to another culture of typhoid bacilli.

13. **In the Liver, Spleen, and Blood.**—Guarnieri[2] reports a case of angiocholitis, produced by the typhoid bacillus, without the characteristic lesions of typhoid in the intestine. The bacillus was cultivated from the liver and spleen, and from the blood of the patient twelve days before death.

The following additional references to cases showing the presence of typhoid bacilli in various organs, but without typhoid lesions in the intestine, have been kindly furnished me by Dr. Flexner:

14. **In the Intestine.** Widal[3] gives the following [illegible]. In a case of Pick's[4] the sero-diagnosis

[illegible] Gen. di

[illegible] May,

[illegible]

had been positive during life. At the autopsy the lesions of typhoid fever were not found in the intestine or in the spleen, but the typhoid bacillus was cultivated from the intestine.

15. **In the Spleen, Lungs, and Pleural Effusion.**—In the case of Chambon and Ménard,[1] the disease presented during life the symptoms of typhoid fever and acute tuberculosis. The sero-diagnosis was positive three times. The autopsy showed granules (tubercles?) in the lungs, meninges, kidneys, liver, spleen, and intestines, but no typhoid lesions in the intestines. Cultures from the spleen, lungs, and pleural effusion gave a bacillus which was identified as the bacillus typhosus.

Flexner[2] and Hodenpyl accept the case of Du Cazal as proved from the bacteriological point of view, but reject other cases as not differentiating the colon and the typhoid bacilli with sufficient accuracy by the methods employed. I do not feel myself competent to decide such a technical point, but will only call attention to two facts:

First, the cases of Cheadle and Hodenpyl seem to have been most carefully studied by the later methods, and that of Kummer and Tavel with equal care by older methods, and would seem to be fairly conclusive. Moreover, if the single case of Du Cazal is accepted, the *principle* is admitted; and if admitted in one case,—rare, it is true, but accepted as certain,—then there is no reason why it may not be true in other cases.

Secondly, while in this or that case the conclusion

[1] Soc. Méd. des Hôp., 1897, No. 2.
[2] Jour. Path. and Bacteriol., 1895, 230.

might be erroneous, it is hard to believe that over a dozen observers—careful men, trained to their work—would *all* be wrong. Even if they did work with imperfect methods, their conclusions, when supported by the three or four acknowledged to have been investigated by accurate and modern methods, it seems to me, must be accepted as in general correct.

Possibly the explanation of those cases of infection in which the typhoid bacilli have attacked not the intestine but other organs may be found in the investigations of Remlinger and Schneider.[1] These authors found the typhoid bacillus in earth, in drinking water, and in the intestinal canal in healthy persons. The bacteria obtained resembled those from true typhoid stools in form, culture, and biological tests, but they were less virulent and the cultures were not affected by the serum-test. They believe that Eberth's bacillus is, to a certain degree, ubiquitous, that it occurs in drinking water, in the soil, and in the digestive tract of healthy persons as well as in those suffering from typhoid; that it can remain in the bowel of healthy persons innocuous until an accidental weakening of the intestine or the appearance of other bacteria gives it the power to manifest its action.

Pancini,[2] after investigating six cases (abscess of the liver from dysentery, suppurating echinococcus cysts, etc.), in all of which a bacillus in all respects resembling the typhoid bacillus was found, but without any typhoid fever or typhoid intestinal lesions, says that one of three conclusions is inevitable: (1) That typhoid bacilli are so frequently found in

[1] Ann. de l'Institut Pasteur, vol. xi, p. 1.

[2] Riforma Med., 1893, 95, in Centralbl. f. Bakteriol., 1893, xiv, 497.

the human intestine as to be regarded as normal; (2) that the colon bacilli can not be distinguished from the typhoid bacilli; or (3) that the typhoid bacilli must be only varieties of the colon bacilli.

Which of these conclusions is correct I do not feel myself competent to judge, and most probably it is as yet an unsolved problem.

Frangulea,[1] in a paper at the recent Moscow Congress, accepts the last very frankly.

Hiss, in an important recent paper,[2] has pointed out the means of differentiating the two with approximate certainty. Flexner[3] has done the same.

[1] Verein's Beilage z. Deutsch. med. Wochen., Sept. 16, 1897, 187.
[2] Jour. of Exp. Med., ii, 677.
[3] Jour. Path. and Bacteriol., 1895.

CHAPTER III.

TYPHOID GANGRENE.[1]

ENGLISH and American authors in the past have not given the attention to gangrene after typhoid which its importance deserves. Thus Reynolds and Bartholow make no allusion to it; Wilson only alludes to gangrene of the lung and the mouth, two of its rarer sequels; Flint mentions it, but had never seen a case; while Murchison in his classical work, and Hutchinson in an excellent chapter on typhoid in Pepper's System of Medicine give it only a brief notice. Nor have the surgeons done it any better justice. Neither Gross, Agnew, Ashhurst, nor Holmes[2] mention it. Barwell[3] refers to it briefly. It is not mentioned in Dennis' or Park's System of Surgery and not alluded to in Treves' System.

We owe our chief knowledge of the subject to French authors and to a few recent German and American publications. Larrey,[4] Hildebrand,[5] Alibert and Labbe mention sporadic cases of gangrene, but attention was first seriously called to its [illegible] typhoid fever in 1857 by Bourgeois[6]

[illegible] notice, but is chiefly an ex- [illegible] Some paragraphs in that [illegible] others have been [illegible]

[illegible] of Surgery. [illegible] 1865. [illegible] 1864, 530.

and Bourguet.[1] The next papers of any importance were by Gigon in 1861 and 1863.[2] The former established the fact of gangrene from arterial obstruction by autopsy, though he regarded the gangrene as a coincidence rather than a consequence of the fever. In 1863, Patry[3] confirmed these earlier observations. The cases reported by these authors were included in the summary published in the Toner Lecture (1876) already alluded to.

Since my own contribution, some of the more important papers are those of Spillman,[4] Gaston David,[5] Barié,[6] Deschamps,[7] Haushalter,[8] Flexner,[9] and Quervain,[10] besides a very large number of individual cases, which have been reported by various authors.

Under gangrene I exclude from consideration the familiar and frequent bedsores, which, though actually often partaking of this character, are scarcely in the same category as the cases I shall consider.

One would suppose *a priori* that gangrene would only follow severe attacks, but so large a number of cases of gangrene have been reported after relatively mild attacks, that we must concede the possi-

[1] Gaz. Hebdom., 1857, 646. [2] L'Union Méd.

[3] Arch. Gén., 1863, i, 129, 549.

[4] Gangrène des Organes Génitaux de la Femme, Arch. Gén., 1881, 7th series, vi, 150.

[5] La Gangrène Typhoide, Thèse de Paris, 1883.

[6] L'Artérite aiguë consec. à la Fièvre Typhoide, Rev. de Méd., 1884, iv, No. 1.

[7] L'Artérite aiguë dans la Cours de la Fièvre Typhoide, Thèse de Paris, 1886.

[8] Merc. Méd., September 20, 1893, 453.

[9] Johns Hopkins Hosp. Rep., Nov., 1894, 120; and Jour. Path. and Bacteriol., Nov., 1894, iii, 202.

[10] Centralbl. f. inner. Med., August 17, 1895, 793.

bility of gangrene in mild cases as well as severe ones. Hence the watchfulness of the physician should never relax by reason of the fact that the case is running a mild course, and that gangrene is an infrequent result of typhoid.

While gangrene is an important complication or sequel of typhoid, it is fortunately rare, so that most practitioners, and even some men of vast experience in large hospitals, have never seen a case—for example, Flint and Murchison. Hölscher,[1] in 2000 fatal cases of typhoid, does not report a single case, though he records 59 cases of thrombosis of the femoral vein, and Bettke,[2] in 1420 cases, found only four cases of gangrene, all limited to the toes. In my former lecture I tabulated 43 cases from typhoid and 56 from typhus.[3] Since 1876, Dr. Westcott has found 90 cases of actual gangrene, of which 72 followed typhoid, in addition to which he has tabulated 21 cases of arterial and 48 of venous thrombosis not followed by gangrene. In its infrequency, therefore, it is in marked contrast to the bone lesions, of which he has found 168 cases, all after typhoid, to which, from my former lecture, are to be added 37 after typhoid and four after typhus, or a total in typhoid alone of 205 cases of bone lesions to 133 of gangrene.

Date of Onset.—While gangrene is generally a late complication during the course of the fever, or an early sequel during convalescence, it is never a very

[1] Münch. med. Wochen., 1891, xxxviii, 43. [2] Inaug.-Diss., Basel, 1870.

[3] Thirty-four of these after typhus were reported by Estlander (Langenbeck's Archiv, 1870, p. 453) in a frightful epidemic following a financial crisis and a series of bad harvests in 1862–67, in Finland.

late sequel, as is the case in the bone lesions. The latter often do not occur until several weeks, sometimes months and occasionally even years, after the attack of fever. This is doubtless due to the fact that the bacilli of typhoid find a favorable nidus in the bones, especially in the marrow, and have been repeatedly demonstrated by stain and culture after six, twelve, or eighteen months, and even after so extraordinary long a period as six[1] or seven years.[2] In addition to this, the slowness of all pathological processes in the hard osseous tissues, as contrasted with their rapidity in the soft parts, would naturally lead us to expect that gangrene would occur far earlier than lesions in the osseous tissues, yet abscess of the brain may occur two or three months after the fever (see Chapter VIII).

The earliest time at which gangrene occurred, I find, is on the fourteenth day,[3] and the latest in the seventh week.[4] By far the commonest time for this dangerous complication to appear is the second and third weeks, during which 39.2 per cent. of all the cases occurred.

The causes for the appearance of gangrene in the second or third weeks, or late rather than early in the disease, are probably twofold. First, during the earlier stages of the disease the general vitality of the patient and the resistance of the tissues are such that they can combat successfully the evil tendencies of the fever; but, secondly and chiefly, just as for

[1] Sultan, Deutsch. med. Wochen., 1894, 675.

[2] Buschke, Fortschritte d. Med., 1894, 573.

[3] Donald, Lancet, 1892, i, 417.

[4] Forgues, Rec. de Méd. Mil., 1880, 3d series, xxxvi, 386.

the production of the intestinal lesions, so for the gangrene, a certain length of time is required for the diffusion of the bacilli and their toxic products, and for their resulting evil effects. Both causes unite in working together and to the same end. By the second or the third week the bacilli and their toxic products have become diffused through the system; excessive feebleness has followed the small amount of food taken and the exhaustion from the continued high temperature; the heart[1] has become weakened, which favors the formation of thrombi, not only in the heart but also in the vessels, either as a result of arteritis or of autochthonous thrombosis; emboli frequently result; and with the sluggish circulation, the general enfeeblement both of mind and body, and the frequently obstructed vessels, the advent of gangrene at this period of the fever should occasion no surprise. Indeed, the surprise is rather that it is so rare.

Let us now consider the pathology, symptomatology, and treatment.

Pathology.—Various writers have been the partizans of one or another single cause for the occurrence of gangrene during and after typhoid fever. This seems to me an error, for, as I hope to show, there are a number of causes, of which one will exist in one case and another in another case, sometimes singly, sometimes in combination.

[1] We do not appreciate how much a continued high temperature alone exhausts a patient. Were the body composed of water alone, to raise the temperature of a person weighing 150 pounds from 98.5° to 103.5°,—*i. e.* five degrees (to say nothing of the expenditure of force needful to keep it there),—requires an expenditure of force equal to raising 285 tons one foot (150×5 = 772 foot pounds). A girl of 100 pounds weight, simply lying still in bed, suffering from such a fever, does daily the work of two or three men.

In my Toner Lecture I was disposed to regard the causes of gangrene as chiefly three: first, the altered blood; secondly, the weakened heart; and, thirdly, the mechanical difficulties in carrying on the circulation, especially in distant parts; and that all of these caused the gangrene by the production of thrombi, either macroscopic or microscopic. Since that lecture was delivered, however, the bacillus of typhoid has been discovered, and has been proved by a number of careful examinations to play an important, and in some cases at least a direct, rôle in the production of gangrene. It is greatly to be regretted that only very few cases have been studied with the scientific precision which they deserve. By calling renewed attention to the subject, however, I hope to stimulate others, especially in this country, to make thorough bacteriological examinations in the future. Every chapter in this monograph shows how deficient our knowledge is for want of such investigations. All the more are such careful examinations necessary, since the opportunities to make them are so rare, and when they do occur it is only too seldom that it is in the hands of men with the opportunity, the capability, and the disposition for making such examinations.

In two cases in my tables ergot had been freely given in consequence of hemorrhage.[1] Were these the only cases of gangrene, one might suppose that this had had a determining influence, but as gangrene followed in 88 other cases, in none of which

[1] Finlayson, Amer. Jour. Med. Sci., March, 1891, and Sarda, Rev. Gén. de Clin. et de Thérap., 1892, vi, 401.

had this drug been administered, its exhibition must be considered as merely incidental.

As a foundation for our study of the pathology of typhoid gangrene, let me recall a few of the facts which have been demonstrated bacteriologically.

First, not a few cases of typhoid fever suffer from a mixed infection. This is much more apt to lead, however, to other disorders than gangrene. For example, as I shall show hereafter, there have been a number of cases of tetanus, erysipelas, anthrax, and malignant edema, due, of course, to a mixed infection of the typhoid and these specific bacteria (Chapter XIX). In addition to this, the large number of cases of suppurative disorders in various organs, bones, spleen, muscles, etc., presume the presence of the ordinary pyogenic bacteria, and their presence has been proved by stain and culture. In two cases in my tables pyemia or septicemia, a rare condition, is noted,[1] in both of which the pyemia followed a phlebitis without gangrene.

Turning now to the cases in which bacteriological examinations have shown pure cultures of the bacillus of Eberth, we must note, as bearing upon the occurrence of gangrene, that they may be found: (*a*) In the blood; (*b*) in the endocardium; (*c*) in the walls of the arteries; (*d*) in the walls of the veins; (*e*) in the thrombi; and (*f*) in the perivascular tissues.

(*a*) **Typhoid Bacilli in the Blood.**—This has already been fully considered in Chapter II, page 23, and need not be repeated.

[1] Wagner, Brit. Med. Jour., 1891, i, 18; and Spillman, Merc. Méd., 1895, No. 13, 145.

(*b*) **In the Endocardium.**[1]—Viti[2] not only found the bacillus of Eberth in the granulations of endocarditis, but by the injections of the bacillus alone into rabbits he was able to produce endocarditis with vegetations.

Vincent[3] records another case occurring in a soldier who was undoubtedly free from a preceding endocarditis, but died from typhoid, and in the vegetations on the mitral valve pure cultures of the bacillus of Eberth were found. Girode[4] made a similar observation. Gilbert and Lion[5] were also able to produce such endocardial vegetations experimentally by injections of pure culture of the typhoid germs.

Besides the actual discovery of the bacillus of Eberth in the endocardial vegetations, it is not uncommon to find ante-mortem clots in the cavities of the heart. Forgues,[6] Beaumanoir,[7] Fritz,[8] and Vallette (quoted by Ferrand[9]) have all recorded such post-mortem findings. These clots are formed probably during the period of cardiac weakness, especially in the second and third weeks,[10] and, as the heart begins to regain its force and lose its frequency, are washed into the circulation as emboli. In the viscera their presence is shown by multiple infarcts; in the legs, by the occurrence of gangrene.

[1] *Cf.* Flexner, Jour. Path. and Bacteriol., 1894, iii, 202, for several cases.

[2] Atti della R. Accademia del Fisiocritici di Siena, 4th series, vol. ii, fasc. 5, 6, 1890.

[3] Merc. Méd., Feb. 17, 1892, 73.

[4] Comptes Rendus Soc. Biol., 1889, 622.

[5] Comptes Rendus, 1889.

[6] Réc. de Mém. de Méd. Mil., 1880, 3d series, xxxvi, 386.

[7] Prog. Méd., 1891, ix, 364.

[8] Charité Annalen, vi, 169.

[9] Contrib. à l'Etude de la gangrène des membres pendant la cours de la Fièvre Typhoïde, Thèse de Paris, 1890.

[10] Drewitt, Lancet, 1890, ii, 1023.

(*c*) **In the Walls of the Arteries.**—Rattone[1] reports four cases in which, in sections of the arterial tunics, he was able to obtain pure cultures of the bacillus of typhoid.

(*d*) **In the Walls of the Veins.**—Haushalter[2] found the bacillus in sections through the veins; and both he and Vaques found the pyogenic microbes in the walls of the veins in cases of typhoid phlegmasia.

Arteritis, endarteritis and peri-arteritis, phlebitis and periphlebitis, have been described by a number of authors, especially Ferrand, Deschamps,[3] Mettler,[4] Barié,[5] Quervain,[6] and Haushalter.[7] With the exception of the last two, the descriptions are pathological, but without bacteriological confirmation. Ferrand[8] quotes numerous cases of endarteritis of the iliac, femoral, and popliteal arteries, followed by thrombosis and gangrene. Barié describes two forms of arteritis; first, an obliterating form, and, secondly, a parietal form. In the first there is profound alteration of the middle coat, the muscular fiber cells being infiltrated with embryonic cells, with sclerosis of the external coat and vegetations in the lumen. Sometimes, indeed, the three coats are indistinguishable. In others the lining membrane is covered with small elevations consisting of masses of round and fusiform cells. There is a loss of elasticity in the vascular walls, which become friable and easily distended. The loss of smoothness

[1] Della Arterite Tifosa in Déhu, *loc. cit.*

[2] Merc. Méd., Sept. 20, 1893, 453. [3] Thèse de Paris, 1886.

[4] Phila. Med. Times, Feb. 10, 1887, 339, and N. Y. Med. Jour., March 0, 1895, 280. [5] Rev. de Méd., 1884, iv, 7.

[6] Centralbl. f. inner. Med., Aug. 17, 1895, 793.

[7] Merc. Méd., Sept. 20, 1893, 453. [8] Thèse de Paris, 1890.

PLATE I.

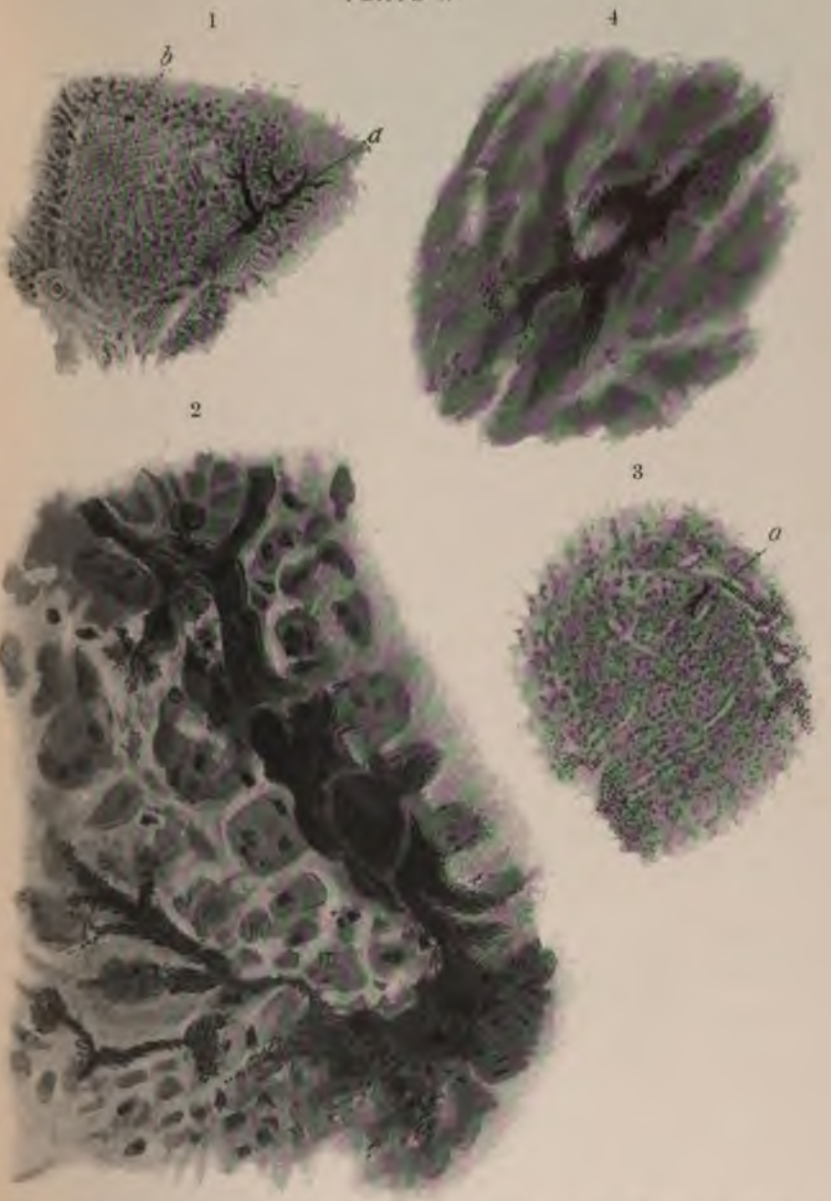


Fig. 1.—Section of liver of a patient dead of typhoid fever. Bacilli were found also in the spleen. *a*, A vessel and its branches completely obstructed by typhoid bacilli; below it and to the left a close small-cell infiltration which almost entirely obscures the vessel. *b*, A so-called lymphoma.

Fig. 2.—Enlargement of Fig. 1, *a*, showing the individual bacilli.

Fig. 3.—The liver of a rabbit dead after an injection of pure culture of typhoid bacilli in the ear veins. Bacilli were found also in the spleen and the mesenteric glands. *a*, A vessel entirely obstructed by typhoid bacilli. The section shows at various points diffused small-cell infiltration.

Fig. 4.—Enlargement of Fig. 3, *a*, showing the individual bacilli. (Fraenkel and Simmonds, "Die etiologische Bedeutung des Typhusbacillen," Leipzig, L. Voss, 1886. Reproduced by the kind permission of the authors and publisher.)

of the intima, the irregularities of its surface, and the diminished caliber from the swelling are readily conceived causes for the formation of thrombi, resulting frequently in gangrene. The thrombi, at first red, but later decolorized, become adherent to the wall, and finally the artery becomes a solid cord. Distal secondary thrombosis is slow. The lumen of the vessel and of its branches is gradually obliterated, and the gangrene, therefore, is correspondingly slow, and not sudden as in embolism; but from the original thrombi secondary emboli may form, and so hasten the gangrene by sudden obliteration of the anastomotic circulation.

As a rule, therefore, in arterial thrombosis the gangrene is dry, but occasionally during the course of the dry gangrene from arterial obstruction, the vein also becomes obstructed, and part or all of the limb may fall into sudden ruin from moist gangrene.

The parietal arteritis, according to Barié, is not attended with thrombosis, and is usually followed by recovery.

That arteritis should occur in typhoid is rendered also probable by its appearance in other allied specific diseases, such as small-pox, diphtheria, tuberculosis, syphilis, rheumatism, etc.

How the bacilli reach the walls of an artery or vein is a question. Ordinarily they are not found in the blood; and yet the fact that they may be widely distributed throughout the body, and that the only reasonable mode of such an extensive diffusion is by the blood, the cases of Flexner, Vincent, and others amply prove (*cf.* Chapter II). Haushalter believes that during their maximum they may reach

the vessels by the vasa vasorum,[1] which, it must be remembered, especially in the veins, reach the middle, and often the internal coat. He is inclined, however, to believe that the process is as follows: that an infection of the pelvic or crural ganglia occurs, followed by an infection of the perivenous cellular tissue by retrograde lymphatic circulation. In these cases he supposes that a periphlebitis exists, followed by a secondary endophlebitis precipitating a thrombus by the ferment furnished by the bacilli. This, I confess, seems to me much less likely than the former view.

(*e*) **In the Thrombi.**—Both Rattone and Haushalter found the bacillus of typhoid in thrombi. The latter calls attention to the fact that he was not able to stain them in the thrombus, due, he thinks, to the fibrin, which, being decolorized with great difficulty, probably obscured the bacilli; but he was able to demonstrate their presence by cultures. He found the endothelium of the veins destroyed. On the surface of the clot next the vein wall a layer of leucocytes was intimately united both to the clot and to the wall of the vein. The typhoid bacillus existed only in the thrombosed portion of the vessels; and he is of the opinion that either the bacilli or their products caused the destruction of the endothelium and the resulting clot, and that, in all probability, in the products of the bacilli was found the ferment necessary to produce the coagulation.

Plate I, from Fraenkel and Simmonds,[2] shows

[1] For a number of cases in which bacteria were found in the vasa vasorum, see Lockwood, Traumatic Infection, London, 1896.

[2] Die ætiologische Bedeutung des Typhusbacillen, Leipzig, 1886.

excellently the actually observed thrombosis of the vessels of the liver by the bacilli of typhoid.

(*f*) **In the Perivascular Tissue.**—Quervain found the bacillus in pure culture in the pus surrounding the popliteal artery and vein. That the bacillus should cause suppuration around a vessel and be found in pure culture ought not now to astonish us.

The pyogenic power of the typhoid bacillus is fully considered in the introduction.

Having now determined the bacteriological facts, let us see how they may be applied pathologically in explaining the causation of gangrene.

The cause of gangrene may be stated in practically a single phrase—*obstruction to the circulation.*[1] The three factors I have already quoted from my first lecture—the altered blood, the weakened heart, and the mechanical difficulties of the circulation in distant parts, especially the last two—still hold good, but there must be added to them the important rôle of the typhoid bacillus in assisting, and often, it may be, in directly precipitating, the coagulation of the blood, which is the cause of the obstruction. Four different varieties of obstruction, therefore, may exist and sometimes co-exist: First, arterial emboli of cardiac origin; secondly, autochthonous thrombi in the arteries; thirdly, autochthonous thrombi in the veins; and, fourthly,—probably, though I believe there have been no cases absolutely demonstrated pathologically,—thrombi in the small peripheral vessels. In Chapter VIII, on the cerebral complications of typhoid, I have reproduced the illustrations of Ohlmacher showing obliterative endarteritis in

[1] *Cf.* Oliver, Lancet, 1896, i, 1778.

suppurative typhoid meningitis. These observations, together with the demonstration of actual thrombi caused by the typhoid bacilli themselves in the vessels as shown by Fraenkel and Simmonds (Plate I), throw an important light, it may be, on this cause of gangrene.

1. **Arterial Embolism of Cardiac Origin.**—This has been observed not only at postmortems, but clinically. Thus, Hayem[1] observed the alteration of the heart two days before gangrene of both legs commenced, the first symptoms being acute pain in the legs with a sensation of cold. The pulsation disappeared first in the dorsalis pedis, then in the popliteal, then in the femoral. Amputation showed that, though the arterial walls appeared to be healthy, the femoral artery was partly obstructed by a clot. The obstruction being only partial allowed a feeble circulation to go on. The popliteal and all its branches below the inferior articular were entirely free from any clot. The patient died, and the necropsy showed endocarditis and clots in the heart; the aorta was obstructed by a clot extending from its bifurcation to a point above the origin of the inferior mesenteric, but the iliac arteries were entirely free. The spleen and the kidneys presented multiple infarcts. Mercier[2] reports also a case of dry gangrene of both legs with fibrinous clots in both the primitive iliacs, the deep femoral and the popliteal, the walls of which were healthy, and in the left auricle there were old fibrinous clots with endocarditis. This form of arterial obstruction is quite common in my table, as will be observed in the résumé on page 74. It leads,

[1] Prog. Méd., 1875. [2] Arch. Gén., 7th series, 1878, vol. ii, 402.

as would naturally be supposed, almost always to dry gangrene, because it eventually cuts off the supply of blood; as a rule, absolutely; though in a few cases, as in the one quoted from Hayem, a small amount of blood may still reach the distal parts and so restrict the extent of the gangrene. The very facts, also, that the walls of the arteries were healthy and that there were multiple infarcts in the viscera, testify to the cardiac origin of such emboli.

In addition to this I have called attention on pages 76–78 to the remarkable difference between arterial and venous obstruction in relation to their distribution to the right or left sides of the body. The almost even balance in arterial obstruction and the great inequality in venous obstruction may point also to the heart as a factor in arterial obstruction.

2. **Arterial Thrombosis.**—In a great number of cases in the table there were no evidences of preceding cardiac disease, and yet obstructive clots were found in the arteries, and were followed by dry gangrene. These are undoubtedly autochthonous thrombi, probably produced largely from the ferment furnished by the bacilli themselves, or possibly more frequently from an endarteritis, such as has been already described.

The consequences of the thrombosis, as of the embolism of the arteries, will be a greater or less degree of dry gangrene, the extent of which will depend upon the completeness or incompleteness of the obstruction.

Sudden thrombosis may be precipitated by very slight causes—*e.g.*, Morax[1] records the case of a man

[1] Bull. Soc. Méd. Suisse, 1867, quoted by Mercier.

of thirty-five who, when convalescent, after a walk felt sudden severe pain in his left leg, followed by gangrene from arterial thrombosis. Sometimes, even after a careful post-mortem, it may be impossible to determine the origin of the obstruction.

Thus, Valette (quoted by Ferrand) relates a case of occlusion of the iliac arteries, gangrene of the right leg, vulva, and perineum, with bedsores on the thigh and sacral regions setting in on the forty-second day. The leg was amputated after eight weeks, and the patient, age eighteen, died twenty-three days later. The necropsy showed red hepatization of the right lung. There was neither myocarditis, endocarditis, nor arteritis, but in the left ventricle was a clot as large as a small egg, yellow, hard, and elastic, and extending to the semilunar and the mitral valves: and at the iliac bifurcation on the right side, a whitish-gray clot obliterated the external and internal iliacs.

In the Toner Lecture (see Appendix, Fig. 1), I have copied a figure from Meusel, in which gangrene (necrosis) of a large portion of the skull resulted from a clot in the middle meningeal. As the patient recovered and no mention is made of any cardiac trouble, the clot was probably an autochthonous thrombus.

3. **Venous Thrombosis.**—This is much more frequent than the arterial form, probably from the more sluggish circulation, in addition to the infectious processes which undoubtedly sometimes cause a phlebitis or a periphlebitis.

The phlebitis very frequently results in phlegmasia alba dolens. Usually this passes away in a

relatively short time, but it may be the cause of very persistent trouble and annoyance, as in the following instance:

Case I.—E. C., Milford, Del., age forty-two, first consulted me July 9, 1897, at the instance of Dr. Marshall. He is an exceedingly large, stout man, who, six years ago, had an attack of typhoid fever. During his convalescence phlegmasia developed in the left leg, from which he has suffered ever since. The leg is still swollen, especially from the knee down, is very hard, tense, and brawny, is covered with crusts of exudation, and is subject constantly to small furuncles and the formation of small ulcers and occasionally of distinct abscesses. One of these, above the internal malleolus, was opened about six months ago and has never healed. At this site there is a small crater, one cm. in diameter, going through the entire thickness of the skin to the muscular tissues, and the seat of an exceedingly foul discharge. The itching of the limb also annoys him very much. I directed him to foment the leg sufficiently to take all the crusts off, and then to apply an ointment of resorcin, 30 grs. to the ounce. Under this treatment he rapidly improved, and was soon entirely well.

Venous thrombosis results in gangrene in a moderate number of cases; but in the majority, as in venous thrombosis from puerperal fever, pneumonia, etc., gangrene is much less apt to follow venous than arterial thrombosis. The circulation is not nearly so completely cut off by venous obstruction as by arterial, since the collateral venous channels are less frequently blocked. Moreover, in arterial obstruction the limb below the obstruction is entirely deprived of blood, whereas in venous obstruc-

tion the blood is dammed up in the part beyond the obstruction. The circulation may be hindered, but if it be not practically entirely arrested, a feeble nourishment goes on, sufficient at least to prevent gangrene.

It is to be observed that both in venous and arterial thrombosis, especially the latter, the clots are often discontinuous. (See cases of Hayem, Mercier, and Beaumanoir.) This, possibly, may be due to isolated spots of local infection from the bacilli.

The venous clots are often very extensive, much more so than the arterial, as shown by a number of cases in my table. Thus, in a case of de Santi,[1] the clot extended downward to the deep femoral vein and upward through the common iliac to the vena cava. Beaumanoir[2] reports a case in which there were not only clots in the arteries of both legs, but also fibrinous clots in the right ventricle and pulmonary artery and its branches, in the left auricle, and in the femoral arteries and veins and in the aorta to the level of the first intercostal artery. Naturally such extensive obstruction was followed by gangrene of both lower extremities. Clots extending into the vena cava are also reported by Dumontpallier,[3] Sorel,[4] and Bouley.[5] These were all fatal, but Mackintosh,[6] reports one followed by recovery. The attack of typhoid was followed by scarlatina, but the swelling of the legs was noticed

[1] Rec. Mém. de Méd. Militaires, 3d series, vol. xxxv, 1879, 502.
[2] Prog. Méd., 1891, ix, 364.
[3] Comptes Rendus Soc. Biol., 1879, 6th series, vol. iv, parts 2 and 3.
[4] L'Union Méd., 1882, 3d series, vol. xxxiv, 521.
[5] Prog. Méd., 1880, viii, 998. [6] Glasgow Med. Jour., 1892, xxviii, 54.

before the latter. The collateral circulation was re-established by dilatation of the internal mammary, superficial epigastric, external pudic, internal saphenous, and circumflex iliac veins on both sides. The diagnosis seems, therefore, fairly well established.

Occasionally, as would be supposed, venous thrombosis is followed by sudden death, as in a case reported by Nauwerck,[1] of thrombosis of the left iliac vein, which was followed by sudden embolism of the pulmonary artery while the patient was at stool, and death occurred in ten minutes; and in another reported by Bouley,[2] also of thrombosis of the external iliac vein, which extended to the inferior vena cava and the right auricle of the heart, and the patient died from syncope. As would naturally be supposed also, the thrombosis, both arterial and venous, but especially the former, is apt to lead not only frequently to double gangrene,—for example, of both lower extremities,—but sometimes to a gangrene which is so symmetrical as to remind one of cases of Raynaud's disease. Richard[3] has reported two such cases in brothers. A curious coincidence is seen in the case of two sisters reported by Trélat,[4] both of whom had double cataract after typhoid. They were both young (one twenty-five). At least one was successfully operated on. Had the bacillus of Eberth then been known, it would be interesting to know whether the cataract was due to a local invasion.

[1] Correspondenzbl. schweiz. Aerzte, 1879, 485.
[2] Prog. Méd., 1880, viii. 908.
[3] L'Union Méd., 1880, 3d series, xxix, 1025.
[4] Gaz. des Hôp., 1870, lii, 417.

When the gangrene results from venous obstruction rather than arterial, as a rule it is moist.

Not uncommonly thrombosis of the arteries and veins is either successive or simultaneous. In either case, the gangrene is apt to be a combination of dry and moist gangrene. Occasionally, when the venous thrombosis follows the arterial, the gangrene will be at first of the dry variety in the distal parts and when the venous obstruction occurs moist gangrene will follow in the proximal.

4. **Thrombosis in the Peripheral Vessels.**—In addition to the three forms above recited, there are a number of cases reported in which the disease began as dry gangrene in the toes and gradually crept up the leg. The persistence of pulsation in the dorsalis pedis and other higher arteries showed that there was no arterial thrombosis or embolism in the arteries higher up, but after a time the coagulation, which presumably had begun at the periphery, extended centrally, and first the dorsalis pedis, then the tibials at the ankle, and later the popliteal and even the femoral were successively obstructed, resulting, of course, in a more wide-spread gangrene. The symptoms show that none of the three preceding conditions existed, but they enable us by analogy to reach the conclusion that spontaneous thrombi formed in the distal vessels. Whether the cause of this thrombosis is a bacillary infection or not has not been studied with that care which it deserves, and no absolute pathological or bacteriological confirmation of this view, I believe, has been reported.

Symptoms of Gangrene. — The symptoms of gangrene are marked and characteristic. Let us

suppose the case to be one of arterial embolism or thrombosis. Toward the end of the fever, especially in the third week or early in convalescence, as weakness is giving place to strength and the brightest hopes of speedy recovery are cherished, sudden, severe, and persistent pain is felt. This may be at the seat of the impending gangrene, though perhaps more commonly it is in the obstructed artery, especially in the femoral, popliteal, or tibial, and radiates thence toward the periphery. It is followed by numbness, coldness, loss of sensation, and sometimes of motion, and in a short time discoloration and all the other usual evidences of gangrene appear. Sometimes, but not usually, these local symptoms precede the pain. If the vessels of the foot or at the ankle, or even the popliteal, be examined, the pulsation will be found feeble or utterly extinguished, while higher up, at the seat of the obstruction, the artery will be changed into a moderately firm but very tender cord, in which we may sometimes differentiate the obstructed artery from the non-obstructed vein—an important point in prognosis. Week by week, sometimes day by day, the growth of the secondary coagulum may be traced upward by the progressive abolition of the arterial pulsation and by the upward march of the gangrene. If old cicatrices from burns or unhealed eczematous ulcers, old fractures, or varicose veins exist, all *loci minoris resistentiæ*, they will be among the earliest parts to yield. Blebs may form in the earlier stages, but generally they will dry up and the parts will mummify, although, as already indicated, moist gangrene may supervene if a large clot form much higher up, or if, in addition

to the artery, the vein also becomes extensively obliterated, thus involving great masses of moist tissue, such as the thigh, in sudden ruin.

As is generally seen in cases of dry gangrene, days or weeks will elapse, if the patient live so long, during which nature, as usual, makes a powerful effort to rid herself of the dead parts by the establishment of a line of demarcation. On the establishment of this, the pain often ceases.

In case the primary obstruction is in the vein and it becomes obliterated extensively and completely by a thrombus, or a simultaneous venous and arterial thrombosis occurs, then the gangrene will be of the moist instead of the dry variety. It will present the usual appearance of moist gangrene. The vessels at the seat of the obstruction will be very tender and can be felt as hard cords. The clot will extend sometimes rapidly and widely, and the gangrene will be more extensive in the area involved and far more acute in its disastrous clinical course, as would naturally be expected.

In the variety of gangrene beginning in the peripheral vessels, the symptoms will vary somewhat. It is not so uniformly in the lower extremity, and is much more frequently symmetrical. If small in extent, pain is not apt to be a leading feature. The onset is often earlier, and from the nature of the case its progress is more acute and its limits more quickly defined; so that usually, within a few days at least, the boundary of the gangrene is well defined. Its area also is usually much less than in those cases in which a perceptible coagulum exists, not often extending in the leg beyond the foot or ankle; and

if it occur in the nose, ears, genitals, etc., it rarely involves surrounding parts to a large extent. Sometimes, however, it may extend more widely, as in a case of typhus and starvation, mentioned by Lyons,[1] in which the patient walked to the workhouse, and on baring his chest the whole of the right side was "a dark, olive-green, jelly-like, tremulous mass." The abdominal wall is sometimes similarly involved. The probably irregular area in which the stasis of the blood will take place in this form also accounts for the great irregularity generally seen in the line of demarcation; whereas, if a well-defined thrombus exists in a large vessel, the gangrene is apt to be fairly evenly bounded. This sudden history is usually followed by a speedily decided issue. Death follows quickly, or reaction and recovery set in within a short time, instead of long hanging in the balance.

In my Toner Lecture I collected in all 113 cases of gangrene, to which Dr. Westcott has added 90, making 203 in all. Excluding from the former collection 34 cases following typhus reported by Estlander in Finland, as they were the result of special and local causes (see Toner Lecture), of the remaining 169 cases 115 followed typhoid fever and 40 typhus fever. Some of the latter were undoubtedly really typhoid. In the following résumé I shall combine the results of the former and the latter series together.

The influence of *age* is not very marked. Of 140 cases, there were under fifteen, 26 cases; from fifteen to twenty-five, 64 cases; after twenty-five, 50 cases.

[1] On Fever, 191.

This will not differ much from the normal age-distribution of typhoid.

But *sex* seems to have a marked determining influence. Of 155 cases, there were, males, 90; females, 65,—or about three males to two females.

The *site of the gangrene* is more striking than either age or sex. It attacked the

Ears in	6 cases.	Anus in	5 cases.
Nose in	10 "	Genitals in . . .	20 "
Face, neck, and trunk in	47 "	Legs in	126 "

That is, of 214 cases in which the location is stated, in 151 it was in the lower extremities, genitals, and anus, and in 16 more in such peripheral districts of the vascular system as the ears and the nose.

I have found in the two series 128 cases of venous coagula following typhus and typhoid, especially the latter, in which the site is stated. Only four cases involved the upper extremity alone, two of which were followed by gangrene. Two involved both arm and leg, but all the other 124 cases were limited to the lower extremities. Gangrene of both venous and arterial origin (including both thrombosis and embolism) form most frequently during or just after the period of greatest cardiac weakness, a weakness felt most at such distant points as the legs. Of 41 arterial cases, 18; and of 107 venous cases, 40, occurred in the second and third weeks of the fever —that is to say, of 148 cases 58 (39.2 per cent.) occurred in the second and third weeks.

These figures, it seems to me, are most instruc-

tive. In discussing the pathology, I gave marked prominence to the sluggish peripheral circulation as a mechanical factor in the production of the gangrene. Even though we admit in many cases the determining influence of the bacilli of typhoid, the striking clinical fact above established must be explained by any accepted pathology. Moreover, this fact is still further emphasized when we consider the cases involving the joints (Chapter IV), of which 70 were in the lower extremities as against 17 in the upper; and those involving the bones (Chapter V), of which 112 were in the lower extremities as against 41 in the upper.

If arteritis or phlebitis or emboli of cardiac origin, whether bacillary or not, were the sole or even the preponderating cause, then gangrene certainly should attack the upper extremities, the head, the neck, and trunk with far greater frequency than is indicated by these statistics. Just as in gangrene from other causes, often of non-bacterial origin, such as ordinary senile and diabetic gangrene; or of probable bacterial origin, as in scarlet fever, measles, and the other exanthemata, the legs suffer so much more than all the other parts of the body put together, so in typhoid gangrene the familiar rule holds good. It is also in the lower extremities that venous thrombi causing phlegmasia alba dolens are most frequent in other diseases of microbic origin, as in puerperal fever, pneumonia, septicemia, pyemia, and even in tuberculosis and malarial fever.

In both these classes of diseases arising from half a dozen or more different bacteria and in those entirely apart from any bacterial influence, the one

striking fact is that *the legs suffer far more frequently than all other parts of the body put together.* It is, it seems to me then, good common sense and good pathological sense to seek for the efficient, the exciting, the actual cause determining the location of the obstruction and the frequent gangrene in the legs themselves *as legs;* that is, as distal parts of the circulatory system.

The distribution as to *left- and right-sided* gangrene is very striking. In the early series I did not make such a differentiation, but in the present series of 90 cases I have found that of 46 cases of arterial gangrene, there occurred on

Both sides,	8 cases.
Right side,	19 "
Left side,	19 "

showing an exactly even balance of the two sides.

In the veins, however, the facts are strikingly different. Here the obstruction involved

Both sides,	4 cases.
Right side,	10 "
Left side,	38 "
Total,	52 cases.

This, as we know, is in accordance with the usual experience in other diseases. Why the left side should be so much more subject to gangrene due to venous obstruction, as also to phlegmasia alba dolens, than the right side, has been a subject of speculation for many years. My own conviction is that the obstruction to the return of the venous blood, by reason of the compression of the left common iliac

vein where it passes under the right common iliac artery, is the most potent factor; slight in itself, it is true, but when the blood is in unstable equilibrium between fluidity and coagulation, this slight retardation is in most cases just sufficient to precipitate the coagulation upon the left rather than upon the right side.

The same predominance of the left over the right side holds good in cases of venous obstruction, and the same balance of the two sides in cases of arterial obstruction, when they are *not* followed by gangrene. Excluding three involving the Sylvian artery,[1] all of which occurred upon the left side, producing right hemiplegia,[2] and one of the right brachial artery,[3] and two of the pulmonary artery,[4] the 15 cases of *arterial* thrombosis without gangrene involved both sides in four cases; right side in six cases; left side in five cases.

Of 47 cases of *venous* thrombosis without gangrene there were:

Bilateral, ...	3 cases.
Right side, ...	13 "
Left side, ...	31 "
Total, ...	47 cases.

That is to say, combining together the cases of

[1] Huguenin, Correspondenzbl. schweiz. Aerzte, 1879, No. 15, 449, in which there was also a tubercular meningitis; Barberet and Chouet, Gaz. Hebdom., 1879, 2d series, xvi, 329; Vulpian, Rev. de Méd., 1884, iv, 162.

[2] For a later important contribution to this rare sequel of typhoid see Osler, Recent Studies in Typhoid Fever, Johns Hopkins Hospital Reports, vol. v., and in the Journal of Nervous and Mental Disease, May 1896, p. 295.

[3] Sumner, Boston Med. and Surg. Jour., 1883, cviii, 200.

[4] Nauwerck, Correspondenzbl. schweiz. Aerzte, 1870, 485, and Mayet, Ann. de la Soc. de Méd. de Lyon, xxviii, 2d series, 1880, p. 111.

venous obstruction, whether followed by gangrene or not, of 99 cases (52 + 47) there were:

Bilateral,	7 cases.
Right side,	23 "
Left side,	69 "

While in 61 cases (46 + 15) of *arterial* obstruction there were:

Bilateral,	12 cases.
Right side,	25 "
Left side,	24 "

This extraordinarily even distribution in cases of arterial obstruction would seem to suggest that the cause is much more frequently embolic than has been hitherto believed, whereas in the venous obstruction the immense preponderance is on the left side and is probably due to the cause mentioned on page 77.

The *male genitals* suffer very rarely as compared with the relative frequency in which the genitals in women suffer. Dr. Westcott, after a most extensive search, only found one case[1] in which the scrotum and the feet both suffered from gangrene. A few cases were noted in my earlier lecture. Barring the organic destruction, no special result follows, except possibly hemorrhage, for one case is recorded of death from a hemorrhage of ℥xxx from the scrotum.[2]

The *female genital organs*, however, suffer not infrequently, probably from the neglected condition of many of the patients and the constant soiling of the parts, as a result of unconscious and unavoidable discharges, especially in women. I have found in all 21 cases, of which 17 followed typhoid and four

[1] Tunis, Univ. Med. Mag., 1889–90, ii, 195. [2] Murchison, p. 194.

followed typhus. Fifteen cases were in young persons from seventeen to twenty-seven, except one child of five and five women of thirty-two years of age and over. In 18 of these cases there was gangrene of the labia extending sometimes to the perineum and the thigh, and even to the lumbar region and up the back, as in a case reported by Spillmann.[1]

The disorder manifests itself either as a distinct gangrene of the external genitals or by gangrenous ulcers forming in the vagina. The former occasionally is followed by complete closure of the vagina and retention of the menstrual flow, as in a case reported by Guéneau de Mussey,[2] in which an operation resulted fatally, and in another, reported by Martin,[3] in which sloughing of the upper vagina and the entire cervix uteri occurred.

The vaginal ulcers appear usually on the posterior wall and lead occasionally to recto-vaginal fistula. One is reported by Lebert,[4] in which, during convalescence in the seventh week, chill, fever, and diarrhea set in, and four weeks later the fistula was discovered by injection. It was situated in front of the hymen and was as large as a five centime piece. A month later she died of pelvic peritonitis. A second is reported by Liebermeister.[5] It was caused by the sloughing of a large piece of the recto-vaginal septum, in mass. The large fistula thus produced healed without operation. A third case of vesico-vaginal fistula and gangrene of the vulva appears in the present table reported by

[1] Arch. Gén., Feb. and March, 1881. [2] Gaz. Hebdom., 1867, 652.

[3] Centralb. f. Gynekol., 1881. [4] Anat. Pathol., ii, 307, and Pl. cxv.

[5] Ziemssen's Cyc., Amer. ed., vol. i, 184.

Schick,[1] and in the fourth, my own case, both recto-vaginal and vesico-vaginal fistulæ occurred.

A résumé of this unique case, of which I quote the earlier part from my former lecture, is as follows:

Case II.—Mrs. M. D. was under my observation in St. Mary's Hospital from 1873 to 1876, and is the only case I have found of both recto-vaginal and vesico-vaginal fistulæ. Up to March, 1872, she was perfectly healthy, when, at the age of thirty-four, she had a severe attack of typhoid fever for four months following exhaustive nursing during her husband's fatal illness also from typhoid. About the fourth week the labia minora sloughed away to a large extent, and both urine and feces escaped by the vagina. In October, 1872, she was admitted to the hospital, under the care of my colleague, Dr. Grove, with two large vesical openings (separated by a slight bridge of tissue), which had destroyed the posterior part of the urethra and the floor of the bladder up to the uterus, and one rectal opening an inch in diameter, and 1½ inches above the anus. Dr. Grove operated on her three times unsuccessfully; once on the rectal opening by the rectum, when he divided the sphincter, and twice by the vagina. From December, 1873, to December, 1875, I did nine operations. Thrice unsuccessfully I attacked the fistulæ proper, when, becoming convinced that the attempt to close them was hopeless, with her entire consent, after a full explanation of the consequences of the operation, I proceeded to close the vagina. At first I attempted to preserve and utilize the remnant of the urethra, which gave me great trouble and necessitated several operations; but at the twelfth operation, December 28, 1875, I gave up the attempt, excised the useless urethra, and closed the entire vulval aperture by ten

[1] Wien. klin. Wochen., 1892, vi, 413.

silver sutures. The operation was a complete success. At the time of the delivery of the Toner Lecture, nearly seven weeks after the final closure of the vulva, I stated that she defecated, menstruated, and micturated entirely by the rectum, and without the slightest trouble. She rose usually once, sometimes twice, in the night, and micturated only five or six times during the day. My greatest fear was that the feces, softened by the urine, would pass into the vagina or bladder and give trouble, but up to that time at least, none had arisen, and she was happily rid of the annoyance which had continued four years. Soon after this, however, a small fistulous opening appeared in the cicatrix, caused probably by the feces. This healed after a thirteenth operation, and when my lecture was printed (May, 1878) she had remained entirely well for over fifteen months. In the last four operations, instead of the usual sigmoid female catheter to empty the bladder I inserted the curved branch of a pocket-case male catheter into the bladder and the vagina through the anus and the recto-vaginal fistula, thus draining these cavities, while I drained the rectum below the eye of the catheter by an ordinary drainage-tube inserted into the rectum, lest the feces should be softened by the urine and then pass into the vagina. They answered admirably. The difficulty in obtaining a cure, I believe, lay partly in the inherent difficulty of the case and partly in her deteriorated health ever since the fever.

Her later history is as follows:

Menstruation ceased in February, 1887, over eleven years after the closure of the vagina. December 11, 1888, she again came to me complaining of pain in her rectum and vagina, and stated that the urine was intermittent, sometimes escaping and sometimes not. She told me that for the thirteen

years since the last operation she had been absolutely comfortable; that she was only obliged to rise about twice in the night to evacuate the rectum; and that neither the urine nor the menstrual flow, while it had continued, had irritated the rectum, nor, so far as her sensations went, had the feces annoyed her by gaining access to the vagina. By inserting a finger into the rectum, I found that the old fistula between the vagina and the rectum had so contracted that it would barely admit the point of my forefinger. This examination showed at once that a calculus had formed in the vagina, which acted like a ball valve. Of course I readily partly crushed it by means of a pair of curved forceps introduced through the rectum. The portion I secured uncrushed weighed 70 grains and measured ¾ by ⅝ of an inch. She made an entire recovery in three or four days.

On May 7, 1896, she called again to say that while she had been perfectly comfortable for the seven years since the removal of the stone, three weeks before she called a small abscess had formed at the former outlet of the vagina, and that that morning the urine had commenced to dribble away. Examination showed the orifice of the vagina firmly closed excepting at one small point just admitting a probe, through which some urine was escaping. Rectal touch showed that the recto-vaginal fistula was the same as before. I advised her to keep the parts clean and wear a napkin, and wait to see whether the small fistula would not close spontaneously. In two weeks this hoped-for result followed, and she is again entirely relieved of her distressing disability and has continued well up to the end of 1897.

The case is particularly interesting, not only for its unusual character and its cause, but because I believe it was possibly the earliest case in which the urethra itself was entirely removed and the vagina

closed, the rectum thus being made to serve the triple purpose of a reservoir for the urine, the menstrual discharge, and the feces. It is an encouraging fact that in any case requiring similar treatment, the later history shows that for twenty-one years she has only twice had the least trouble, once from a small calculus forming in the vagina and once from a small abscess forming in the cicatrix, which abscess spontaneously closed. Instead of being a constant source of disgust to herself and everybody about her, a hospital patient dependent upon charity, as she could not earn her daily bread, and a Pariah cut off from all society, she has been enabled to become self-supporting as a nurse, and to enter freely into her wonted social relations.

Gangrene of the *perineum or around the anus* arises in a few cases in both sexes. In women it is generally by extension of the process from the vulva. I have notes of nine men and six women, the sex not being given in two other cases. Excepting five cases,—one of ten, two of eighteen, and one each of twenty-one and twenty-two years of age,—they all occurred, when the age is stated, from thirty-nine to seventy-four years of age, later in life than most of the other sequels of typhoid. This is presumably due to the fact that in later life the nutrition of the perineum is apt to be less vigorous than in early life. Typhoid was the cause in 14; typhus in three. Excepting one in the second week, they all occurred also rather later than other cases of gangrene; namely, from the third to the seventh week—in other words, during distinct convalescence; and to this is probably due the fact that 11 recovered and five died, one from

hemorrhage by sloughing into the rectum. In a number of cases the bones of the pelvis were involved as well as the soft parts, and this may have been the real origin of the trouble. This also probably partially accounts for the later occurrence of these cases.

Most of the perineal cases resulted in perineal fistulæ. Three fistulæ were caused by necrosis of the pelvic bones or sacrum and nine by gangrenous ulcers, which sloughed not only externally, but in five certainly communicated with the rectum, and probably did so in others. The only case of primary invasion of the anal region I have found was communicated to me by Dr. Betz, of Oakville, Pa., and well illustrates the sudden and extensive ravages which typhoid gangrene may produce. There can be little doubt, I take it, that such a case resulted from extensive thrombosis of the vessels supplying the tissues which sloughed. That the upper part of the rectum itself was saved is due, I suppose, to the escape of the main trunk of the hemorrhoidal arteries from participating in the thrombotic process.

Case III.—"H. L., age ten, fell ill with typhoid fever August 1, 1890. The remainder of the household, consisting of his father, mother, a younger sister, and two servants, a man and a woman, soon were all victims of the same disease. Presumably there must have been some serious local cause. The boy's temperature rose as high as 105°, but nothing out of the common occurred until the end of the fifth week, when he complained of irritation around the anus, which parts were found to be only discolored. Within twelve hours after attention was thus first called to them the tissues in the ischio-rectal fossa separated from the surrounding tissues and dis-

charged. The rectum was relaxed and protruded. It was gangrenous and speedily separated some distance above the sphincters. Retraction then took place, and a formidable cavity was left. Urination, strange to say, was not interfered with. The wound healed by granulation quite rapidly under a dressing of lint saturated with carbolized oil, dusted with iodoform. Fecal evacuations were involuntary, of course, but the greatest cleanliness was enforced. An apparatus was needful to retain the feces at first, but was laid aside by December. During recovery regular dilatation of the anus was made lest cicatricial contraction should produce obstruction. His recovery was absolutely complete, not only as to general health, but as to control of the evacuations."

Occasionally gangrene attacks very unusual regions or organs. Thus single cases are reported of gangrenous suppuration of the gland of Bartholin (Spillmann[1]); of the tongue, with cyanosis of the face and great dyspnea (Gaston David[2]); of the uvula (Freudenberger[3]); of both ears after ergot, ʒvj, had been given in six doses (Sanda[4]); of the lips, in which a secondary staphylococcus septicemia, ensuing probably from carious teeth, destroyed life (Spillmann[5]). The cheeks are attacked more frequently, and noma or cancrum oris is noted in my table as having been observed nine times, and, as usual, is very fatal, five of the nine having succumbed, the result in one being unrecorded. The lungs also suf-

[1] Arch, Gén., March, 1881.

[2] Quelques Consid. sur la Gangrène Typhoide, Thèse de Paris, 1883.

[3] Aertzlich. Intelligenzbl., 1880, xxvii, 7.

[4] Rev. Gén. de Clin. et de Thérap., 1892, vi, 401.
Merc. Méd., 1895, No. 13, 145.

fered from gangrene in five cases, of which three died. As to all of these, there is nothing peculiar calling for more than their mention as indicating the protean manifestations of typhoid gangrene.

I append the following case of sloughing of the face, which if not one of noma was at least akin to it. The notes were kindly sent me by Dr. J. N. Hall, of Denver, Colorado:

Case IV.—"Hattie J., American, eight years old, together with a brother and younger sister had severe typhoid in 1889. Course of disease normal until thirteenth day, when considerable swelling of genitals occurred, but disappeared after a few days. On sixteenth day small bedsore on back, and two or three more on nineteenth day. Extensive passive congestion of lungs. On twenty-ninth day temperature 103°, 'swelling of right sublingual gland' (as I have it noted), 'feeling like the parotid in mumps.' On thirty-third day discharge of pus from right ear, with appearance of black slough, size of five-cent piece, on right cheek, opposite right first lower molar. On thirty-fifth day this was one by two inches, and on thirty-sixth extended to corner of mouth. Severe hemorrhage (arterial) occurred on this day and on thirty-eighth day patient died."

Treatment.—To the treatment which I advocated twenty years ago little can be added. The preventive treatment, such as good food, fresh air, the best hygienic surroundings is the most important. Should the heart flag, its action must be maintained at all hazards; alcohol in liberal doses is perhaps the best remedy. Digitalis, strychnin, spartein, strophanthus, and other cardiac tonics of the later pharmacopeia may be added. The body should be carefully

examined, especially those parts of it which experience has shown likely to be attacked, pre-eminently the lower extremities, and in women, and especially girls, the genitals. The arms, neck, and head being exposed are much more likely to attract attention should they be attacked by gangrene than those which are covered by the bed-clothes (*cf.* Chapter IV). If baths are used, care should be taken that no mechanical injuries are inflicted, especially on the legs. Chapman's ice- and hot-water bags, alternate heat and cold, with very moderate friction and stimulating liniments, should be advised and the use of the constant current as a means of stimulating the collateral circulation, both in the deep as well as the superficial parts, will be of service. If gangrene is not only threatened, but actually sets in, the gangrenous parts should be kept as free as possible from infection by the use of antiseptic dressings.

The question of operation naturally is one of the most important that is raised. In gangrene of the genitals, head, neck, or trunk, operation, of course, is limited to the removal of the dead and sloughing tissues and especially in the promotion of the utmost cleanliness, particularly in parts of the body soiled by urine, feces, or the menstrual discharge. Detergent washes and stimulating douches, the keeping of the rectum free from accumulated feces, and thorough and free incision of abscesses in the vicinity of the anus are to be especially commended.

In the extremities, if amputation is necessary the time when it shall be done depends largely upon whether the gangrene arises from distinct obstruc-

tion by a palpable thrombus or embolus, or whether it arises in the peripheral vessels without such an appreciable mechanical obstruction of the main vessels. In the latter case, the line of demarcation is usually established quite early, and the disease is generally unlikely to advance beyond this line. Amputation, therefore, should be done as soon as the line of demarcation is well pronounced, and it may be done but little above this line, since there is no obstruction in the vessels higher up which would threaten the integrity of the flaps.

In the cases where a distinct thrombus or an embolus has formed, however, the obstruction is very apt to extend farther and farther, as time goes on, by secondary thrombosis. At what level, therefore, the limit between the tissues which must necessarily die and those in which nature can still keep up a healthy life will occur, can not be stated definitely until the line of demarcation is well established. In a very few cases, even when gangrene seems inevitable, the patient may yet recover without gangrene. Two such cases are reported by Phillips[1] and Salles.[2]

But the facts obtained by a study of my two series of cases aid us very distinctly in deciding the level at which amputation should be done. When the clot extends only up to the popliteal, the leg may escape gangrene altogether; and, should it follow, I have found it limited, in 21 cases, to the foot six times, to the lower half of the leg once, and to the upper calf in 14 cases. When the clot extended into the femoral, the gangrene extended to the upper calf in 11 cases and to the thigh in eight cases.

[1] Lancet, 1891, i, 1207. [2] Lyon Méd., 1893, No. 3.

When the clot extended above Poupart's ligament the gangrene was limited, in 15 cases: to the foot in one, to the calf in eight, and extended above the knee in six. Amputation in these cases, therefore, should not be done, as a rule, until a well-defined and probably final line of demarcation has been formed. When operating, the leg should be made bloodless by elevation and kept so by very careful digital compression. The Esmarch bandage, as pointed out by Quervain, should not be used, partly because it may injure the vessels of the stump and so favor a new arterial or venous thrombus, and partly because it may break up an existing venous thrombus and give rise to a dangerous embolus. To this I would add another evident objection: that the septic fluids in the tissues should not be forced into the general circulation. The hemorrhage will be slight, since certainly the artery, and often both the artery and the vein, will be obstructed, so that the "muscles will look like meat soaked in salt and water, and there will be no oozing from the marrow of the bone."[1]

Quervain's method of operating was both ingenious and useful. After forming an anterior flap, and before making the posterior flap containing the vessels of the lower thigh, he only disarticulated the bones at the knee joint; next he dissected the femur loose for 12 cm. above the joint, and divided the bone; then exposed the vessels and ligated them; and last of all formed his posterior flap. The wisdom of ligating the artery before dividing it was shown by the fact that in the amputated part

[1] Drewitt, Lancet, 1890, ii, 1023.

it was found to be filled with a loose clot, which would almost certainly have been dislodged by the manipulation if the flap had been made prior to ligation, and so have caused considerable hemorrhage. Such patients have not a drop of blood to spare.

As a general rule, therefore, it is best to wait for the line of demarcation, but the operation should not be deferred long after its appearance. If danger of septic infection or speedy exhaustion appear, amputate immediately at or above the probable limitation of the disease. The extension of the disease, if the femoral be free, will not be, in the majority of cases, above the tubercle of the tibia. If the femoral be involved, necessitating an amputation of the thigh, the resources and the safety of modern antiseptic surgery would lead us, in general, to amputate; but in some cases it may be a serious question whether expectant treatment and a relatively long subsequent amputation might not be less dangerous than an earlier operation. In two cases[1] amputation of both legs was followed by recovery.

[1] Butler, N. Y. Med. Rec., Sept. 28, 1889, 342; and Durand, Arch. de Méd. et de Pharm. Mil., July, 1894.

CHAPTER IV.

TYPHOID AFFECTIONS OF THE JOINTS.

Rheumatic Typhoid Arthritis.—Besides typhoid arthritis, properly so called,—which I shall shortly consider at length,—there are two other forms of arthritis which occasionally, but far less frequently, are connected with typhoid fever—viz., a rheumatic and a septic form of arthritis. While, like the usual typhoid arthritis, both may affect a single joint, more frequently they are polyarticular.

Of the rheumatic variety, in the later series of cases, the case of Despaigne,[1] though the history is not entirely clear, was very probably an example. Multiple ankyloses were found after an interval of eighteen months. Three other cases are distinctly stated to be of this variety, and had a previous rheumatic history. Freyhaus' case,[2] in which the knees, ankles, elbows, and wrists were involved, but recovery of all these joints followed, may be fully accepted as of the rheumatic variety. That of Balzer,[3] which proved fatal from a purulent arthritis of the left knee with non-purulent teno-synovitis of the left wrist, may also have been an instance. But this case probably, and still more certainly that of Robin, in spite of the opinion of the author, seems to me to be not so

[1] Lapersonne, Des arthrites infectieuses, p. 120.
[2] Berl. klin. Wochen., 1891, xlii, p. 1.
[3] Robin, Gaz. Méd. de Paris, 1881, No. 40, p. 559.

much rheumatic as septic in character. These, with those of Rendu [1] and that of Ménard,[2] I should class together under the next variety.

Septic Typhoid Arthritis.—In the case of Robin [3] there were purulent arthritis of the little toes and pus in the sheaths of their extensor tendons; purulent arthritis of the knee-joints, of the right ankle, and of the knuckle-joint of the right middle finger, the pus again extending into the sheaths of the tendons of that wrist; purulent arthritis of the right shoulder and of the left elbow, with suppurative periostitis of the left tibia and abscess around the larynx and trachea and the left costal cartilages. Could one draw a more striking picture of a septic case? No other result than death could be expected.

The two other cases, though only involving one joint, seem to have been septic.

In Rendu's case, the patient died from a purulent arthritis of the right hip-joint with abscesses in the thigh and osteomyelitis, and the autopsy disclosed a purulent pleuro-pneumonia and nephritis.

It is much easier now to understand these septic cases than twenty years ago, before bacteriology was born. They result from a mixed infection with the typhoid and the pyogenic bacteria. In the third case this is definitely assigned as the cause, the port of entry being the sacral bedsores. In fact, every case of typhoid fever has a possibility of sepsis, since, apart from the frequent boils and bedsores, the intestinal ulcers are always an open and possible door inviting such infection. Tripier [4] has especially

[1] Quoted by Witzel.
[2] Soc. Méd. des Hôp., Jan. 11, 1878.
[3] Gaz. Méd. de Paris, 1881, 559.
[4] Lyon Méd., 1888, p. 195.

insisted upon the frequent, if not constant, origin of septicemia from the external ulcers.

In Ménard's case it is to be observed that a rare joint—the upper articulation of the sternum—was attacked, the pus soon reaching the anterior mediastinum. Analogous to this one case of the later series are those of Fraentzel [1] and Werner [2] in the earlier series.

A septic arthritis in typhoid runs the usual course of similar septic inflammations, and has their frequently fatal termination in spite of all treatment; all the more so in typhoid since it follows or accompanies so exhausting a disease. There is nothing, in respect either of the symptoms or the treatment, to which I need call attention, except to the necessity for the most vigorous stimulation. But the occasional occurrence of such septic cases and their uniformly fatal result should warn the physician to heed the very first complaint of pain in any part, especially if the complaint be made early in the case. It may be the beginning of a rheumatic or a septic case, which he may possibly be able to carry through safely. If not of either of these varieties, it may be the signal symptom of the usual typhoid arthritis, in which the danger is not of suppuration or even generally of ankylosis, still less of death, but of a totally unexpected complication—viz., dislocation, especially of the hip.

[1] Ein Fall von acuter Mediastinitis in Verlauf eines Ileotyphus, Berlin. klin. Wochen., 1874, xi, 97.

[2] Verbreit. sinuöse Geschwüre auf der Brust, Perfor. vorder Mittelfellraum., Plötzlich. Tod an Verblutung, Typhöse Geschwüre im Darm., Med. Corresp. Württemb. aerztlich. Verein, Stuttgart, 1859, xxix, 76.

Typhoid Arthritis Proper.—(*a*) **Polyarticular Variety.**—Typhoid arthritis proper may affect more than one joint, though the monarticular form, especially in the hip, is far the most frequent and the most serious.

In the second table, excluding cases involving the vertebræ, which are considered later, the polyarticular cases involved: (1) Both the upper and the lower extremities in 3 cases—viz., wrist and knee, 2; elbow and both ankles, 1. (2) The lower extremities alone in 6 cases—viz., both hips, 2; both ankles, 2; hip and knee, 1; hip and ankle, 1. (3) The upper extremities alone in two cases—viz., both wrists, 1; shoulder and elbow, 1.

Combining these together, it will be observed that the lower extremities were involved in nine cases as against five in the upper.

Not one of the cases died; a marked contrast to the six cases of the rheumatic and septic forms, of which four died. Even, therefore, a polyarticular arthritis does not seem to add to the danger of death in typhoid—a most comforting fact to the physician, the patient, and his friends. Even ankylosis is an infrequent result. Beside the case of Despaigne,[1] already considered, in one case ankylosis of both hips followed a faulty posture, persisted in long after the fever.[2] This case recovered motion after forcible flexion under ether. In Clarke's case[3] ankylosis of the elbow persisted, very possibly for want of early passive motion.

[1] Lapersonne, Des Arthrites Infect., p. 120.

[2] Gibney, Trans. Amer. Orthop. Assoc., 1889.

[3] Jour. Amer. Med. Assoc., April, 1891, 473.

In the earlier series three other cases of ankylosis were noted, making six cases of ankylosis out of 84 cases of arthritis in the combined tables.

In its symptoms, save that more than one joint is involved, the polyarticular form of typhoid arthritis does not differ from the monarticular; and as the latter form, especially in the hip, is so important, this will be considered more fully.

(*b*) **The Monarticular Variety.**—The monarticular form of typhoid arthritis affects the larger joints, such as the elbow and shoulder, the ankle and knee, but above all the hip. The pain is usually slight, though sometimes very severe and prolonged. The swelling is generally readily observed in all joints except the hip and the shoulder, where it is probably obscured by the muscular masses about these joints combined with the tardy increase in the swelling. Usually the arthritis arises spontaneously, but occasionally from periostitis or necrosis extending into the joint. Pus rarely forms, and hence suppurative or fistulous openings are rare.

In only one case,[1] in fact, of the 41 cases of the second series was there suppuration in any joint, that case involving the knee. This indisposition to suppuration is well shown in Dunin's case,[2] in which, though the infection was severe enough to produce abscesses of the buttock and hip, many furuncles and suppurative otitis media with arthritis of the right elbow and shoulder, these joints themselves did not suppurate. The result is, therefore, generally a gradual return to usefulness, although in six cases

[1] Tarbox, N. Y. Med. Rec., Aug. 24, 1889, 209.
[2] Deutsch. Arch. f. klin. Med., 1886, xxxix, 369.

I have found ankylosis. Of 84 cases, the lower extremities were affected in 70, the upper in only 17, seven of the cases involving a joint in both; for occasionally two large joints are affected at once. Arthritis, therefore, resembles other surgical febrile affections, such as gangrene, necrosis, etc., in affecting mainly the lower extremities, as do also thrombosis and the ordinary edema.

The frequency of these joint troubles is not very great. Murchison does not even name this complication, nor do any of our text-books, either on Surgery or Practice, except a few lines by Volkmann, in Pitha and Billroth's Handbuch. Güterbock,[1] Hellwig,[2] Parise,[3] and Friedheim[4] are the only authors who have treated them at all fully. In the literature of the last fifty years, which is practically covered by my two series of tables, I have collected in all 84 cases involving the joints. That they are of great importance and demand our utmost attention will be seen at once when we consider that of the 84 cases named spontaneous dislocations occurred in 43; 40 times in the hip, twice in the shoulder, and once in the knee—*i. e., more than one-half of all the cases of typhoid arthritis are followed by spontaneous dislocation, nearly all of which are in the hip-joint.*

Dislocation of the Hip-joint after Typhoid Fever.—These dislocations require more particular notice. They are analogous to the dislocations which

[1] Archiv f. klin. Chir., xvi, 58.

[2] Ueber die Affect. des Hüftgelenks bei Typhus, Marburg, 1856.

[3] Archiv. Gén., 3d series, 1842, xiv, 1.

[4] Ueber die Spontanluxation des Hüftgelenks nach Typhus, Berlin Thesis, 1885.

have been observed in locomotor ataxia, the exanthematous fevers, hemiplegia, sciatica, and rheumatism,[1] as pointed out by Stanley[2] in 1841. In one case[3] it was noted that for three months before the typhoid attack the child had been suffering from ordinary coxalgia.

Unfortunately, there are on record very few examinations of such joints post-mortem, and very few bacteriological examinations of the fluid in the joints, and these are generally either negative or reveal the presence of the pyogenic organisms. The only instances I have found are as follows:

1. A patient of my own, in St. Agnes' Hospital, had an attack of suppurative arthritis of the knee-joint, arising during typhoid fever, which I operated on, and the aureus and albus were found in the cultures made by Dr. Bevan. Unfortunately, the notes of the case have been lost, but the bacteriological examination was the chief point.

2. A case of typhoid fever, under the care of Professor Hare and Dr. Thomas G. Ashton at the Jefferson College Hospital, which I saw with them. She was a girl of sixteen, admitted on January 16, 1897, with a temperature of 105.3°. She made a good recovery and was discharged from the hospital. In the fifth week swelling and some pain were ob-

[1] Spontaneous luxation has been observed after scarlatina by Dittel (Oester. Zeitschr. prakt. Heilk., 1861), Seinton (Rev. d'Orthop., 1892, 354), Kirmisson (Bull. et Mém. Soc. de Chir., 1894, xx, 213), Eisendrath (Annals of Surgery, Oct, 1897, 451); after acute articular rheumatism by Verneuil (Bull. et Mém. Soc. de Chir., 1883, 781), Seinton (*loc. cit.*), and Kirmisson (*loc. cit.*); and after influenza by Eisendrath (*loc. cit.*).

[2] On dislocation, especially of the hip-joint, Med. Chir. Trans., xxiv, 1 See also Malgaigne, Fract. and Disloc., Paris, ii, pp. 218–226, 882–887

[3] Phocas, Gaz. des Hôp., 1894, Nos. 132 and 135.

served in the left leg, the swelling extending from above the knee to the ankle, but she recovered from this without any especial trouble. Soon afterward, however, the swelling returned, and was accompanied by severe pain and great tenderness, especially in the knee-joint, so that motion was impossible. I saw her on April 22, 1897, and again on June 18th. On both occasions the knee was aspirated, but the fluid proved to be entirely sterile. The arthritis resulted in ankylosis in marked flexion, which will require later operative treatment.

3. Klemm [1] reports the case of a girl, age fourteen, who was ill with typhoid for fourteen weeks, and who had lain for two months with the knees almost touching the thorax. An iliac dislocation of the left hip-joint resulted. An abscess formed over the trochanter, which, on being opened, gave exit to a reddish turbid fluid, like that of an old hemorrhagic hydrocele. The trochanter was bare and eroded. Facial erysipelas followed, and she died nineteen days after admission. At the post-mortem typical typhoid lesions were found. The capsule of the hip-joint was much distended but without any fluid in it. The fluid of the abscess contained the typhoid bacillus. The hip-joint does not seem to have been examined bacteriologically.

4. Schüller [2] found, in cases of inflammation of the hip- and knee-joints after typhoid, "in the serous fluid only round cocci and some streptococci, and single small bacilli resembling those which are found not infrequently on the intestinal surface, to which atten-

[1] Arch. f. klin. Chir., 1893, xlvi, 862.

[2] Witzel's Gelenk- und Knochenentzünd., p. 52. (No reference is given.)

tion was first called by Klebs, but which, however, Koch, Gaffky, and others do not regard as absolutely characteristic of typhoid. The bacilli now regarded as the typhoid bacilli,—the ovoid form first described by Eberth,—I could not find in the contents of the joint. In another case of typhoid which I had to examine during life, I could not find any micro-organisms."

5. Danlos and Strauss[1] report the case of a man of twenty-nine whose fever, even at the beginning, was accompanied by pain and swelling in the knees and elbows, and on the fifth day the patellæ were "floating" from the effusion; soon purpuric spots appeared but no taches rouges. He died on the eighteenth day, his articular disease not having materially changed. The day before his death the right knee was punctured, the fluid found being apparently clear synovia. At the necropsy the lesions of typhoid were found. The right knee-joint was healthy; cultures from the fluid in the joint and of the blood were sterile.

6. Orloff[2] injected cultures into joints in dogs and rabbits, and reported that this was followed by swelling in twenty-four hours, with hemorrhage in the synovial membranes. A thick, tenacious, turbid fluid was produced in the joints, which later became more distinctly purulent. Microscopically, there were found pus-corpuscles, and in the earlier days after the injection, typhoid bacilli.

This last result may possibly be the explanation of the impossibility of finding the bacilli in the joint

[1] Soc. Méd. des Hôp., 1887, 3d series, iv, 35.

[2] Centralbl. f. Bakteriol., 1890, 366.

affections in man, at least in some cases. Having done their malign work, they may have disappeared.

Grancher,[1] however, obtained from a teno-synovitis the typhoid bacillus.

The result in these five cases in man and one series of experiments in animals must leave us in doubt as to the rôle of the bacillus of Eberth in the joint affections of typhoid. The future must afford us the data for a certain conclusion. The need for such bacteriological examinations, at the best very rare, is most evident.

It is to be hoped that this hiatus in our knowledge will be filled before long, especially as it is so easy to obtain the fluid by aspiration, an operation in itself desirable to prevent dislocation. But though we are still largely ignorant of the bacteriology of the joint effusions, yet from the analogy of the other tissues to which I have alluded it is probable that the bacillus will be found to have invaded the joints also, and possibly later to have died out. The irritation caused by the bacilli or their toxins will readily account for the slow but steady accumulation of fluid in the joint. Parise[2] has shown experimentally that artificial distention of the hip-joint will produce a posterior luxation. That the capsular ligament will stretch more readily from the slowly increasing distention of disease, as compared with the rapid distention in experimental cases, is evident. I well remember the astonishment and incredulity I felt twenty years ago, when I first encountered a case of dislocation of the hip-joint alleged to be due

[1] Bull. Méd., 1892, vi, 1271.

[2] Arch. Gén., 3d series, tome xiv, p. 1.

to typhoid fever. But incredulity had to change to belief, as I found case after case in undoubted relation to the fever as a cause, and the more than two score cases now collected leave no room to doubt either the fact or the pathological explanation.

The *cause* and *symptoms* of the trouble may now be considered at some length.

Usually in the period of convalescence following, therefore, the prolonged exhaustion, there arises a subacute synovitis, with a gradual serous distention of the capsular ligament, which, having reached a certain point, may slowly subside, and no further evil follow. In five cases, however, this burst externally, producing sinuses, but in only two of them was the discharge purulent. In both of these there was also necrosis of the bones. The main result is a slow, generally unperceived, elongation of the ligaments,—*e. g.*, of the hip,—with perhaps also a swelling of the so-called gland at the bottom of the acetabulum. This distention will spend its force mainly posteriorly, since the inverted Y-ligament reinforces the capsular ligament in front. Given this condition, the slightest force will dislocate the head of the femur, usually upward and backward on the dorsum of the ilium. In one case a fall to the floor produced it, in three others turning over in bed, and twice the lifting of the patient in the arms from one bed to another. But in all the other 34 cases no cause was assignable, and it is, therefore, likely that it was mere muscular contraction, which becomes more vigorous as health gradually returns—the very time when these dislocations most often occur. In one of the shoulder cases a subcoracoid luxation was

caused by the patient's assuming the erect posture. Gravity had here probably some influence, together with the muscular exertion. The dislocation of the knee was also posterior, the result doubtless of the muscular traction of the hamstrings.

Typhoid was noted as the preceding fever in 15 and typhus in 7 of the hip cases of the earlier series. In the later series typhoid is the sole preceding fever in all 13 cases of dislocation. *Sex* has not a very marked predisposing influence, for of 35 cases 19 were males and 16 females. The *age* at which they occur is much more noteworthy: 22 were under fifteen years, 10 from fifteen to twenty, 1 was thirty, 1 was thirty-five, and 1 was sixty-one years old; that is, *32 out of 35 were under twenty years old.* The analogy to coxalgia, it will be observed, is, therefore, very marked. Usually they were single dislocations, 12 being on the right side and 10 on the left; but in 3 cases dislocation of both hips occurred.

From the apathetic condition of the patient in some cases, the subacute nature of the lesion, the absence or slightness of the pain, or in some cases its great severity which precludes any movements and therefore any thorough examination, the masking of the swelling by even the wasted muscles about the joint, and, above all, the want of knowledge of any danger or even any possibility of the dislocation, and therefore the neglect to examine the parts thoroughly, it is not surprising that this threatening evil should have been often unobserved. In 16,—that is, nearly one-half—of the cases it is distinctly stated that the *actual dislocation* was the first fact observed, and in most of the others this is probably true.

The date at which the dislocation was, at least, observed was generally after the third week. One case occurred in the first week, 4 in the second, 4 in the third, and 17 in the fourth week or later—that is, during distinct convalescence. Pain was experienced in at least 25 cases, and probably in more. Usually it was not severe, nor was it always strictly localized in the hip, but sometimes extended to the entire leg. In only 3 cases was it referred to the knee, thus differing markedly from the well-known coxalgic knee-pain. Swelling is only distinctly stated in 14 cases, though probably present here as in other joints, but either unobserved or often unrecorded in the brief statements I have found. The variety of the dislocation is not named in 12, but as in all the other 28 it was iliac, there is good reason to believe that this is probably practically always the case. One case, however, is recorded of an obturator dislocation.[1] Shortening is recorded in 18 cases, and where the amount is named was generally 1½ to two inches. In 6 cases the rotation was inward, in 2 outward, and in 2 of the 3 double dislocations both legs were rotated in the same direction,—that is, right or left,—thus producing a peculiar deformity when compared with the apparently reversely rotated body. The head of the bone in 4 cases was freely movable in all directions. This mobility of the head and the singular diversity in the rotation of the limb are additional reasons in favor of the distention theory of its pathology.

As to *treatment*, in consequence of the relaxation

[1] Friedheim, Ueber die Spontanluxationen des Hüftgelenks nach Typhus, etc., Berlin Thesis, 1885.

of the ligaments of the joint, reduction is generally easy when the luxation is discovered early, but if the discovery or treatment be tardy, it is always difficult and often impossible. In 17 cases reduction was successfully accomplished, 13 times by manipulation, twice by extension, and twice by both means. In 11 cases reduction was not effected,—though in one, after extension for a week, two attempts at reduction were made under ether by so good a surgeon as Krönlein,—and in 12 the result is not stated. In the case of obturator dislocation two osteotomies gave a favorable result. In three cases seen late, as progression with a high shoe was quite satisfactory, reduction was not attempted. Only three cases of recurrence of the luxation are noted, a rather surprising fact in view of the relaxation of the distended tissues; but its possibility should be borne in mind and guarded against by the use of a stout binder surrounding the hips or by a plaster-of-Paris spica of the hips and pelvis. No snap is heard on reduction, all tension and suction-power of the joint being lost. Even after reduction the leg may be somewhat longer than the other, owing, probably, to the distention, to the swollen articular gland, and possibly, in old cases, to interstitial changes in the head and neck of the femur and in the acetabulum.

The question of prophylaxis is perhaps the most important of all, and the indications are clear. First, a careful watching and repeated examination of the hip-joint, especially in children, to detect pain or any effusion.

Physicians, having learned that dislocation of the hip is a possible, even though an infrequent, result

of typhoid fever, should heed the first complaint of pain, even in a semi-comatose or delirious patient, particularly if the age be under twenty. Even if the patient does not complain of pain, the hip-joints should be examined in young persons from time to time to detect the first possible sign of effusion, and therefore any danger of dislocation.

If any exist, the position of the leg becomes of the greatest possible importance. As adduction and internal rotation favor spontaneous dislocation, the leg should be kept in abduction and external rotation. The first indication is easily fulfilled by two lateral sand-bags, which may be bridged across in front at intervals by a bandage, to keep the leg at rest, or by lateral splints. The foot may be kept in external rotation by bandages or adhesive plaster fastened to the external sand-bag or splint. Gentle extension by a weight and pulley would be useful in steadying the limb. If the effusion threatens to produce dislocation, aspiration under the strictest antiseptic precautions is a safe and efficient means of prophylaxis. If pus is found,—as is almost never the case,—the joint should be opened and treated according to the existing conditions. In one case[1] the upper epiphysis of the femur became detached, and later was removed. In another,[2] the edge of the acetabulum necrosed.

The two following unpublished cases of dislocation of the hip after typhoid are the only ones I have ever seen personally. For the earlier notes of the first

[1] Rivington, Lancet, 1890, i, 901.

[2] Weil, Prager med. Wochen., 1878, 61.

case I am indebted to her physician, Dr. H. W. Rihl, of Philadelphia.

Case V.—In April, 1891, Miss J., age eighteen, presented herself at my clinic at the Orthopedic Hospital to see if anything could be done to remedy her lameness. Over two years before, on December 14, 1888, at the age of sixteen, she fell ill with typhoid fever and was treated by a homeopathic physician. January 2, 1889, near the end of the third week, she was suddenly taken with two convulsions, when her father, in alarm, sent for Dr. Rihl, who writes me: "It was a well-marked case of typhoid from my first visit, on which occasion she had constant low delirium and sleeplessness, not having slept any for forty-eight hours. It varied from ordinary typhoid in three striking particulars: the convulsions occurring at the end of the third week; aphasia, or perhaps, more precisely, amnesia, and the great pain in the right thigh aggravated and incessant for many days, the origin of which was not suspected until after her recovery. I doubt very much, even if I had understood at the time the cause of the pain, whether I would have been able to apply any apparatus, as her pain was agonizing, and she resisted every touch of the limb. I see a record on my book, of January 11th, 'left knee rigid, painful, and flexed'; on re-reading this, my first impression was that I had made a mistake in writing 'left,' meaning 'right,' but in thinking over the matter I remember distinctly that it was a long time before I discovered the real seat of pain, as in consequence of her amnesia and delirium we could scarcely acquire any information from her until some days after, when she pointed to the right thigh. I have no record of the time of the commencement of the amnesia; it was certainly before January

11th. The fever, as you will perceive by referring to my daily record, was January 12th, 104°, and January 18th, 106°, but these high temperatures continued but a few hours, antipyrin acting very satisfactorily in almost every instance in rapidly reducing the temperature. For so grave a case, the average temperature was remarkably low." By January 11th she could speak a word or two; on the 23d for several days her speech had slowly improved, and by the 30th speech had entirely returned. By February 21st she was able to be up each day, and on May 16th Dr. Rihl ceased his attendance. Meantime the pain in the thigh was at times noted as "very severe," and at other times "less," and it is curious to note that the pain seemed not seldom to increase or diminish with the degree of the fever.

In November, when Dr. Rihl returned from his holiday in Europe, he first found the explanation of the pain in the right thigh, there being an iliac dislocation with marked shortening. No intervening accident had occurred.

When she presented herself at my clinic the dislocation was evident at a glance. The right leg measured, from the internal malleolus to the anterior superior spine, 28 inches, on the left 30¼ inches, but there was practically no difference in the circumference of the calf or the general development of the lame leg. As the dislocation had existed for over two years, and a high shoe fairly well remedied the lameness, I advised that no attempt at reduction should be made.

Case VI.—R. McD., aged sixteen, consulted me November 4, 1897, at the Jefferson College Hospital. She had had typhoid fever in March, 1896. In the third week of the fever she suffered from a great deal of pain in the right hip and a little in the knee, which continued for four weeks. Movement of the leg caused pain in the hip. When she got out of

bed she found she was lame, as the right leg was shorter than the left, and was stiff at the hip, but not at the knee. The shortening I found to be two cm., and the head of the femur was subluxated on the edge of the acetabulum. I made four attempts, under ether, to reduce the dislocation, but finding this impossible I ordered a cork sole for this side.

Case VII.—Dr. Swett, of Connecticut, has kindly given me the notes of a third case of dislocation of the hip. The patient, a woman aged thirty-five, lived ten miles away, so that he was unable to see her often, though the high temperature and extremely active delirium made frequent visits desirable. The nurse called his attention to the pain in the hip at his last visit, in November, 1892. Both he and another physician who saw her three months later, thought at first that it was due to sciatica. The pain arose in the beginning of the sixth week, the second week of convalescence. The dislocation was iliac. Two unsuccessful attempts under ether were made to reduce it.

CHAPTER V.

TYPHOID AFFECTIONS OF THE BONES.

EXCEPTING the laryngeal complications and sequels of typhoid fever, the most frequent are those connected with the bones. In my Toner Lecture I gathered 69 cases. But since then, owing to attention having been called to the subject, the recorded cases have become much more numerous, so that we have collected from the literature of the last twenty years 168 cases, making 237 in all, as a basis of my analysis. Unfortunately, no analysis in the particulars not recorded in the earlier lecture, as printed, can be made, since the detailed tables have not been preserved. This is the less to be regretted, since of the earlier series only 37 of the cases followed typhoid and four followed typhus, whereas the entire number of cases of the second series—168—are from typhoid, making in all 205 cases of all kinds following typhoid. The entire absence in the later table of bone disease following typhus and the almost entire absence of typhus cases in the other surgical sequels, is doubtless partly due to improved sanitary conditions prevailing all over the civilized world, which have well-nigh banished true typhus fever. But it is very largely due also to a more accurate diagnosis of typhoid itself, and a more accurate use of language, thus vindicating what I said twenty years ago, that many of the cases recorded and, therefore,

necessarily tabulated as following "typhus" fever, were really sequels of typhoid or "typhus abdominalis." If, for brevity or through carelessness, the author omitted the adjective, I did not feel at liberty to go behind the record, and, consequently, I was obliged to tabulate it as typhus, though often convinced, from the history and symptoms, that the case was really one of typhoid. To take such liberties with the records of cases would expose one to other possible inaccuracies, and would impair confidence in the accuracy of the later author. It is greatly to be regretted that, especially in Germany, the words "typhus," "typhosus," etc., are still used, instead of "typhoid," or "enteric" to designate the fever.

Pathology.—The causes to which twenty years ago I attributed the necrosis of bone which was at that time by far the most frequently recorded sequel among the bone lesions (50 out of 69 cases), were two: First, thrombosis, or, in some cases, embolism; and, secondly, absolute inanition, or want of nutrition. But modern bacteriology, which did not exist in 1876, has rendered these views almost entirely untenable, and gives the first and by far the most important place to the typhoid bacillus.

It is doubtful whether inanition plays any rôle whatever in producing any of the typhoid diseases of bone, though it is possible that in a patient whose nutrition was at a very low ebb it might lead to necrosis, especially if any injury were inflicted on a subcutaneous bone. The poor nourishment of the bone would be a predisposing, and the injury the exciting, cause of the necrosis.

Thrombosis, or occasionally embolism, can not be

so entirely dismissed as a possible, though most probably a rare, cause of necrosis after typhoid. For example, in the Toner Lecture (Fig. 1) I have given the history and reproduced the illustration of a wide necrosis of the frontal, parietal, and sphenoid bones, traced directly and positively to occlusion of the meningea majora. In the chapter on gangrene (pp. 83 and 86) I have pointed out the fact that, in a number of cases of gangrene in the anal and perineal regions, the bones as well as the soft parts were involved, and that noma or cancrum oris was recorded nine times, of which five, and possibly six, were fatal. One can readily imagine that the gangrene of the soft parts in such a case might easily extend to the bones. This is especially true in cases of gangrene of the lips and cheeks, and, accordingly, among the diseases of bone in the second series we find cases of extensive necrosis of the jaws, often associated with gangrene or sloughing of the cheeks. It is most probable, it seems to me, in such cases that either a single thrombus, or occasionally an embolus, of a main trunk, or more likely a wide-spread vascular arrest in many smaller vessels (as described under Gangrene and under the Cerebral Complications of Typhoid Fever), is the cause alike of the destruction both of the bone and the adjacent soft parts. Thus, in Mears' case[1] there was a sequestrum removed consisting of the greater portion of the body or alveolar process of the upper jaw, with parts of the nasal, malar, and palatine processes, and the teeth as far forward as the canine were lost; in Franklin's case[2] there was

[1] Med. Times, Phila., Aug. 28, 1880, p. 608. [2] Lancet, 1897, i, 553.

necrosis of the whole alveolar process of the left upper jaw, with destruction of the cheek (noma); in Lawton's case,[1] with necrosis of the left upper jaw there was gangrene of the left lower eyelid and cheek, and the orbital contents were all loosened from the bone; and in Alexander's case,[2] with necrosis of the upper jaw there was extensive gangrene of the mucous membrane of the mouth.

In only two cases was the lower jaw involved; one[3] with a sequestrum extending from the canine tooth to the angle of the jaw. In the other,[4] on the twenty-first day circumscribed gangrene of the lung occurred, on the sixty-fifth day parotitis, and on the seventy-first day necrosis of the angle of the lower jaw. In spite of all these catastrophes, the patient, a young man of twenty-six, recovered.

While thus it is possible that the two causes which twenty years ago I deemed most potent still should be deemed to have some influence, the real factor, in which inheres practically the chief power for mischief, is the specific cause of typhoid fever—the bacillus discovered by Eberth in 1880. In the introduction to these studies on the surgery of typhoid fever I have fully discussed the occasional pyogenic property of the bacillus of Eberth—a property which no one, in the light of the most recent researches and experiments, can doubt. In no one of the various complications and sequels under consideration is this pyogenic power more strikingly shown than in the bones. All of the earlier cases,

[1] Lancet, 1879, i, 685. [2] Breslauer aertzlich. Zeitschr., 1887, 271.

[3] Heath, Med. Times and Gaz., Dec. 18, 1869.

[4] Eisenhart, Münch. med. Wochen., 1886, xxxiii, 163.

and many of the 168 cases which we have collected in the later table, occurred before the bacillus of typhoid was known. Its presence has only been sought for occasionally, for the larger number of the cases have occurred in the practice of persons whose taste for knowledge or whose opportunities led them only to record the actual practical facts and not to investigate the scientific causes underlying the facts. There are, therefore, only recorded in the later table 51 cases in which a bacteriological examination was made. In 13 the pyogenic bacteria were found; in all the other 38 the bacillus of Eberth was found. In many cases it was not stated whether it was found in pure culture or not,—and possibly in our table the word "pure" may have been omitted by oversight,—but in 14 of the 38 it is distinctly stated that the bacillus was found in pure culture. In one[1] the colon bacillus was found associated with it,[2] and was responsible for the presence of gas in the pus; and in one[3] the aureus and citreus were also found. In a number of cases bacteriological investigation, at a later date, after operation, showed that a subsequent infection with the ordinary pyogenic bacteria had taken place after the operation.

Still more strange is the fact that in a number of cases with an open sinus for months and even years, and, therefore, one would think, with an almost inevitable ordinary pyogenic infection, there has been found a pure culture of the bacillus of typhoid.

[1] Klemm, Archiv f. klin. Chir., lviii, Heft 4.

[2] Déhu (Etude sur le rôle du bacille d'Eberth dans les complications de le fièvre typhoide, Thèse de Paris, 1893) states that the colon bacillus has never been seen in bone abscess. Klemm's case has been reported since then.

[3] Parsons, Johns Hopkins Hosp. Rep., vol. v.

Thus, Parsons[1] reports a case in which, seven months after the fever began and three months after an incision over the ribs resulting in a persistent sinus, the discharge showed a pure culture of the bacillus of Eberth. In Chapter II, page 19, I have given several other instances, in some of which no added infection by the ordinary pyogenic bacteria occurred, even after years.

It has been suggested that there may have been a mixed infection of the staphylococcus and the typhoid bacillus, of which the pyogenic organism was the active agent in causing the suppuration, but that the staphylococcus had died out, leaving the typhoid bacillus alone.

The experiments of Klemm[2] would seem to support this view. Pure cultures of the typhoid bacillus and the staphylococcus aureus were injected into rabbits, either simultaneously or at an interval of a week. An osteomyelitis was set up in each case, cultures from which showed only the aureus and none of the typhoid organisms.

But the experiments of Déhu,[3] Vincent,[4] Vaillard,[5] and Dmochowski and Janowski,[6] a most painstaking series of observations, especially the last, seem unquestionably to establish the fact that instead of a lesser viability the staphylococcus has a greater; that when mixed with the bacillus of typhoid in a mixed infection the bacillus soon dies, leaving the staphylococcus in possession of the field. If, there-

[1] Johns Hopkins Hosp. Rep., v, 407. [2] Arch. f. klin. Chir., Bd. xlvi.

[3] Le rôle du bacille d'Eberth dans les complications de la fièvre typhoide, Thèse de Paris, 1893. [4] Ann. de l'Institut Pasteur, 1893, vii, 141.

[5] Gruncher, Bull. Med., 1892, vi, 1273.

[6] Ziegler's Beiträge, 1895, xvii, 221.

fore, a pure culture of the typhoid bacillus is found in a suppuration after typhoid, it is very unlikely that the staphylococcus ever existed there.

In marked contrast to its behavior when mixed with the staphylococcus, the bacillus of Eberth and the streptococcus both grow luxuriantly together. But, as I have already shown, the so-constant presence of the bacillus of Eberth in pure culture in the pus from osseous and other lesions in the human subject; the fact that Ebermeier[1] has found a pure culture of the bacillus as early as the tenth and the thirteenth day of the disease (two cases in which the date was too early for the pyogenic cocci to die out); the cases quoted on pages 19 and 26, of long-continued existence of the typhoid bacilli alone in suppurative lesions, and the many experiments referred to on page 42, proving that the bacillus alone does actually produce inflammation and suppuration in bone;—seem to set at rest the theory just alluded to. One positive case in which the typhoid bacillus has actually produced suppuration in animals is worth a dozen in which, in man, the suppuration *may* have been due to a presumed pyogenic organism, which, if it ever existed, has disappeared. The same argument which is used in the chapter on the Cerebral Complications of Typhoid applies here. The cases are too numerous to be all classed as mistakes.

The existence of a mixed infection and a pure typhoid infection are not exclusive the one of the other. Both may be true in different cases, and while the proof is decidedly in favor of the view that suppuration in bone is usually due to the pyogenic

[1] Deutsch. Archiv f. klin. Med., 1889, xliv, 140.

property of the typhoid bacillus, yet there are doubtless cases in which a mixed infection has occurred. After a careful examination of the subject, that is the view of Chantemesse and Widal,[1] who ascribe the osteomyelitis of typhoid to both pure and mixed infections.

That the bones offer a peculiarly inviting field for the ravages of the typhoid bacillus, and especially for its late ravages, often not weeks, but months and years after the attack, has been explained of late by the discovery of two facts: first, the remarkable viability of the typhoid bacilli in general (p. 19); and, secondly, their especial viability in the bones (p. 26).

Being so tenacious of life in the adverse conditions in which their prolonged vitality has been proved, it is not surprising that in the bones themselves, under more favorable conditions, they should be capable of living and doing mischief for a long time.

As long ago as 1873,[2] Pontick noted and described the remarkable changes in the spleen and the bone-marrow in typhoid. After the bacillus of typhoid was discovered in 1880, it was especially sought for in the spleen and in the bone marrow, and it was soon shown that of all the organs these two and the gall bladder are its favorite seats, and that especially in the bones the bacilli exist in the largest numbers and linger the longest.

Quincke,[3] in nine post mortems, found the bacillus eight times in the marrow of the ribs and once in

[1] Bull. et Mem. Soc. Med. des Hop. 1887, 3.

[2] Virchow's Archiv, [illegible] and [illegible].

[3] Berlin. klin. Wochenschr. 1887, No. 43.

the bones of the extremities. Wissokowitsch[1] has called attention to the frequency of the infection of the bone-marrow by the typhoid bacilli, and ascribes to this the frequency of the bone lesions of typhoid. Chantemesse and Widal[2] inoculated rabbits and found the bacilli only in the bone-marrow. Dmochowski and Janowski[3] have shown the same facts.

But besides cases in which the bacilli, though present, had not as yet produced any disease in the bones, there are a large number of cases in which the bacilli have been found in cases of abscess and necrosis of bone, and in periostitis and osteomyelitis, *at very long periods after the original attack of typhoid.* The case of Buschke (p. 20), in which seven years had elapsed and yet a pure culture was found, has already been referred to. The next longest cases are those of Bruni and Sultan (p. 21), in which a pure culture was obtained after six years. A number of similar cases are given in detail on pages 21 and 22.

These instances, showing the accumulation of the bacilli in the bones and their persistence for months, and even years, after the fever, explain the otherwise curious fact that the cases of periostitis, osteomyelitis, necrosis, and abscesses in the bones occur at such long periods after the fever and so much later than most of the other surgical complications and sequels of typhoid. Why in some cases, as in those of Quincke, though the bacilli are present, they do not produce any of these disorders of the bones, we do not know. Probably it is due to the

[1] Dehu, *loc. cit.*, p. 90.
[2] Arch. de Physiol., 1887.
[3] Ziegler's Beiträge, 1895, xvii, 221.

fact that the general health is such that their deleterious tendency and influences are successfully vanquished. Possibly, if not probably, most persons convalescent from typhoid, for a considerable time after the fever have foci of infection which never develop disease, and in time disappear because of returning health and vigor. The cases alluded to suggest, also, the need for care during convalescence and after recovery, lest if the general health suffer or an injury be received, the latent bacilli may be aroused into a dangerous activity and cause disease of the bones. For instance, Mercier[1] relates a case in which six weeks after convalescence the patient fell, striking his fore-arm against a piece of wood. This was followed by periostitis and abscess of the ulna. As this case occurred before the bacillus was known, no bacteriological proof of its typhoid origin was obtained.

These latter cases may be analogous in part to a number of instances in which, after a periostitis has arisen with all the ordinary signs of inflammation, it has subsided, and restoration to health has taken place without the formation of pus, the bacilli being killed or their toxins neutralized, possibly by returning health.

Still another peculiarity of not a few of these typhoid bone cases is that a periostitis will start up, reach a certain point, and then subside, it may be for months, or even years, and then recur, reaching the point where an abscess forms after one or two, or it may be even several, such oscillations.[2]

[1] Rev. Mensuelle de Méd. et de Chir., 1879, iii, 21.

[2] *Cf.* Parsons, Johns Hopkins Hosp. Reports, vol. v.

Two other peculiar clinical features of the bone cases may be explained by the long-persisting and favorable nidus which the bacilli find in the bones. Most of the other surgical disorders of typhoid manifest themselves in a single place—*e. g.*, a monarticular arthritis, gangrene of one leg, etc. While occasionally two joints or both legs may be involved, yet it is rare. In the bones, however, multiple foci of disease are common. Thus, Mercier[1] reports a case in which the left tibia, left scapula, left femur, right humerus, and right tibia were all affected respectively on the seventieth, eighty-eighth, ninety-fifth, ninety-seventh, and one hundred and first days after convalescence began; Péan and Cornil,[2] one involving the left tibia, and, five months later, two places in the other tibia; Park,[3] one involving all the bones of both legs, the pelvis, and the spine; Chantemesse and Widal,[4] one involving the right tibia, left femur, a finger on the left hand, and the left ulna, at eight weeks, eleven weeks, eight weeks, and nearly a year respectively; Hulin,[5] one involving the tibia, the ribs, and the clavicle at the tenth week, nineteen months, and about two years respectively. Caspersohn [6] reports a case involving the left parietal, left tibia, and left ulna, respectively during the course of the fever and three and five months after the fever, with suppurative parotitis on

[1] Rev. Mens. de Méd. et de Chir., 1879, iii, 21.

[2] Bull. Acad. de Méd. Paris, 1891, 3d series, xxv, 599.

[3] Annals of Surgery, 1891, ii, 491.

[4] Bull. et Mém. Soc. Méd. des Hôp., 1893, 3d series, x, 779.

[5] Contrib. a l'Etude des Complic. Osseuses de la Fièvre Typh., Thèse de Paris, 1885.

[6] Festschrift Fr. v. Esmarch, 1893, 455.

the forty-eighth day; Catrin,[1] a case in which the left tibia, left temporal, and the right tibia (twice) were attacked, respectively on the fifty-sixth, sixtieth, sixty-fifth, and eighty-third days; and Fürbringer[2] reports a case in which there were seven successive attacks in the course of four months, involving ten different portions of the body, but followed by complete recovery and disappearance of all evidences of disease. In addition to these cases there are a large number of cases in our tables in which two bones were involved.

Besides this, it will have been observed that in the multiple attacks above referred to the various bones are not involved all at the same time, but successively, and not seldom after long intervals, sometimes even in apparent later health. (*Cf.* case of H. W., p. 123.) So long as the bacilli remain in the bones, so long is the patient in danger. He is only safe when they are totally eradicated, and as to the date when this has happened, we can, apparently, only tell by the cessation of the attacks. The following case illustrates repeated recurrences of periostitis and superficial necrosis prior to an attack of typhoid, and that the fever did not, apparently, cause a renewal of the disease in the bone:

Case VIII.—R. W. P., clergyman, age thirty-one, first consulted me November 5, 1877. At nine years of age, presumably from exposure to cold, he had several attacks of periostitis of the entire left tibia and of the left humerus, the latter only in one spot. In both of these places this was followed by necrosis. He lost several pieces of bone from the tibia at

[1] Gaz. des. Hôp., 1896, No. 42. [2] Brit. Med. Jour., 1890, i, 1033.

various times until he was fifteen years old. At twenty-eight he had another attack of periostitis of the tibia, followed by suppuration which was entirely superficial and was not followed by necrosis. At twenty-five he had typhoid fever, during which he lost flesh very rapidly, he thinks as much as 30 pounds in three days, the cerebral symptoms being the most prominent. He was in bed for a month, but returned to his college duties in two months. No periostitis, necrosis, or any other trouble occurred in the bones after his typhoid fever. About September 1, 1877, six years after the fever, two small spots of periostitis appeared again in the middle of the tibia, and at present (Nov. 5, 1877) there are two points at which the bone is exposed—one ½ of an inch in diameter, the other only a pin's point in size. The bone is dry, red, and tender, but is not painful in walking. The bone later discharged, since which time he has been well.

Park[1] has called attention to the rapid growth which often follows an attack of typhoid in adolescents, sometimes as much as a millimeter a day, and ascribes it to the typhoid irritation of the osteogenetic tissues. The long persistence of the bacilli in the bones may readily explain this, especially if they are present in such small numbers as to produce only a mild irritation but are not numerous enough to cause inflammation and suppuration.

In a few cases the osseous lesions have arisen at the seats of old fractures or strumous cicatrices. Thus, Routier and Terillon[2] report a case of periostitis of the left ulna and right tibia at the seat of previous fractures in childhood, and Finlayson[3] a

[1] Mütter Lectures on Surg. Pathol., 260. [2] Prog. Méd., April 12, 1884.
[3] Amer. Jour. Med. Sci., March, 1891.

case of necrosis of both tibiæ at the site of old strumous lesions marked by a scar.

Recent injury seems to be the cause which has precipitated the osseous lesions in some cases. Thus, Mercier[1] and Levesque[2] report three cases in which the osseous lesions followed a fall after the fever, and Clarke[3] one in which a fall on the knee preceded the fever. As the suppurative periostitis of the lower end of the femur did not occur until the fifth week after defervescence, the relation of the two is doubtful. A case reported by Jackson[4] in a man of forty-two is, curiously enough, attributed to the injury inflicted by the use of the stethoscope. Periostitis of the third rib followed five months after the beginning of the attack, and when he was in complete health he alleged that while in bed he felt soreness at that point from the pressure of the stethoscope.

Colzi's experiments[5] may throw some light upon this phase of the disease. The injection of two c.c. of a pure culture in a rabbit's ear always gave negative results, but if the injection was preceded by a subcutaneous fracture an abscess resulted in 11 out of 14 cases. The well-known development of tubercular "abscesses" after injury would reinforce this view.

Witzel[6] has called attention to the—as he believes—much greater recent frequency of the bone lesions of typhoid, and attributes them to the more frequent

[1] Rev. Mens. de Méd. et de Chir., 1879, iii, 21.

[2] De la périostite dans la convalesc. de la Fièvre Typhoide, Paris, 1879.

[3] Jour. Amer. Med. Assoc., April 4, 1891, 473.

[4] Brit. Med. Jour., 1885, i, 428.

[5] Jordan, Beitr. klin. Chir., 1893, x.

[6] Park's Lect.

injuries received from the sides and edges of the bath-tub, since this method of treatment has been introduced. While this may not be so, yet the cases above cited would seem to show that an injury is quite capable of precipitating such troubles in the bones, and that great care should be taken that in bathing no accidental injury, even a slight one, should be received, and also that injury should be carefully avoided, even long after recovery.

Allied to the influence of injury is that of muscular strain. To illustrate this, I quote from my earlier lecture a case in point, with the history for two years later. It illustrates the wide-spread mischief that may follow in the osseous system, when put to the test by labor, months and even years after such a fever.

Case IX.—H. W., a remarkably stout, healthy lad of sixteen, was attacked Dec. 17, 1871, with typhoid. He was delirious for four weeks, was in bed four months, and first got out of doors in May, 1872. Bed-sores had formed, but were kept in check by incessant care. In the autumn of 1872, not yet being strong, he went to work at riveting in an iron works, which required him to stand and use a ten-pound hammer, the main strain being naturally on the right arm and leg. His right arm soon began to swell, and finally four fistulæ formed. After the removal or discharge of several pieces of bone, this arm recovered in about a year. Returning then to the same work, his health being still impaired, his right thigh began to trouble him, and broke out and healed several times, discharging several pieces of bone. He came under my care in July, 1875. He had then a scar and five open sinuses in the thigh, all leading in the direction of the bone, and in one,

just above the knee, a fragment of dead bone an inch long was found. This sinus and a second just below the patella, an offshoot from it, threatened to invade the knee-joint. Meanwhile, in the fall of 1874, though he had not done any work on account of his right leg, the left thigh broke out, and a sinus in the direction of the bone was established, but no dead bone was ever actually found here. In January, 1875, an abscess also appeared in the left arm, and after the discharge of some bone finally healed. I enlarged all the existing sinuses in the right thigh, removed the dead bone, and, after I had treated the case carefully for four months, all the sinuses healed. A new one, however, appeared early in 1876 in the right thigh, but no dead bone was found. In February, 1876, it healed, broke out again in July, and did not heal until December, after a counter-opening had been made. In July, 1879, periostitis again manifested itself at the insertion of the internal hamstring tendons of the right leg. An incision was made down to the bone, giving exit to pus. Another abscess also developed over the sternum at the origin of the right pectoralis major. This also was opened and the pus evacuated. He had gone to work as an engineer meantime. I have not seen him since. His health markedly improved early in 1875, and since my various operations he has grown to be exceedingly robust and hearty again. His right knee, which was stiff from the sinuses among the muscles of the thigh and near the knee-joint, became as mobile as ever, and he works with ease. The abscesses in the two arms were at or near the deltoid insertion; of those in the right leg, the first was at the insertion of the gluteus maximus, the last at the insertion of the hamstrings, that in the left leg near the lesser trochanter, and one at the origin of the great pectoral muscle, all points at which muscular strain would come in standing and hammering, and later in his work as an engineer.

In this case the recurring attacks of periostitis leading to abscesses and superficial necrosis covered a period of at least eight years, and very possibly continued after I lost sight of him. They involved the right humerus, both femurs, and the sternum in eight or ten successive attacks, and were nearly all at points of muscular attachment, and, therefore, of strain. At the same time, it is to be observed that they were all essentially local attacks, the general health being good. They may easily be explained on the theory that the bacilli being present in the bones (the case was observed before the bacillus of typhoid was known), a local irritation was caused by muscular strain, and the bacilli were re-stimulated to the point of abscess formation, and even of necrosis.

On looking over our tables I find that a considerable number of cases would seem to justify the inference that muscular strain may have been the immediate factor in producing the periostitis or other lesion. Of all places in the body to be attacked by periostitis, such points as the anterior superior spine of the ilium, the tuberosity of the ischium, and the great trochanter of the femur would be the last to be thought of. Indeed, I do not remember myself ever to have seen them involved in any case of ordinary periostitis or in any case I have seen recorded in medical literature. But in our tables I find that Sacchi[1] records a case of osteo-periostitis of both anterior superior spines of the ilium, of the tuberosity of the left ischium, and of the sacral and coccygeal regions, the right-sided troubles developing during

[1] Revista Veneta, Jan., 1889.

convalescence, the left two months later. Now, all of these points are points of attachment of large and powerful muscles of the thighs and hips, which would be among the first called upon for a relatively severe exertion, as with returning health the patient would begin to stand, walk, and work.

Ebermeier[1] records another case involving the tuber ischii and another of the inner side of the head of the tibia (inner hamstrings). Hulin[2] records another case of a suppurative periostitis of the left great trochanter.

Turgis[3] attributes an osteo-periostitis of the humerus eight months after the fever, partly to a blow and partly to excessive muscular action. Martha[4] records a case involving the inner side of the head of the tibia and the inner condyle of the femur, the tibia being so enlarged at the point of disease as to prevent full extension of the knee. The bone disease arose two and a half months after the patient had gone to work, and the points attacked were those of muscular attachment.

Another case, reported by Verchère, quoted by Turgis[5] is curious by reason of the alleged cause, and may be an illustration both of traumatism and muscular strain. A periostitis of the sternum, in a woman of sixty-eight, was attributed to pressure from cutting bread while holding the loaf against the chest. In a large number of other cases there is no

[1] Deutsch. Arch. f. klin. Med., 1889, xliv, 140.

[2] Contrib. à l'Étude des Complic. Osseuses de la Fièvre Typhoide, Thèse de Paris, 1885.

[3] Contrib. à l'Etude de l'Ostéo-périostite Conseq. à la Fièvre Typhoide, Thèse de Paris, 1884.

[4] France Méd., April 4, 1888.

[5] Thèse de Paris, 1884.

doubt that the periostitis developed at points of muscular attachment, but no mention is made of the precise point involved; often only "the humerus," "the thigh," etc., are mentioned, because the attention of the reporter probably had not been called to the possible relation between the disease and muscular strain. The cases described are, however, I think, sufficient to establish the probability, if not the certainty, of the inference. In the shaft of the tibia, however,—the most frequent site,—this reason can not be assigned.

Varieties of Disease of Bone after Typhoid.— The two series of cases show a marked contrast as to the frequency and infrequency of the various forms of osseous lesions reported up to 1876 and after that date.

Disease.	Series of 1876.	Series of 1896.	Total.
Necrosis,	50	35	85
Caries,	12	1	13
Periostitis,	3	107	110
Osteitis (bone abscess,)	—	12	12
Osteomyelitis,	—	10	10
Granuloma,	—	2	2
Exostosis,	—	1	1
Uncertain,	4	—	4
	69	168	237

The table is not absolutely accurate. Many of the cases may overlap—*e. g.*, a periostitis may result in caries or an osteomyelitis in a necrosis. Many of the terms are used loosely by the reporters, who often do not nicely or accurately distinguish between caries and necrosis, caries and osteomyelitis, or osteomyelitis and periostitis. The importance of

osteomyelitis especially has not been recognized by the profession at large until of late years, and its distinctive symptoms, and even its existence, twenty years ago were for the most part unrecognized. In fact, in typhoid bone lesions, as in the same lesions from other causes, the two essential forms of disease of the bone are (1) osteo-periostitis and (2) osteomyelitis; while caries, necrosis, bone abscess, etc., according to various circumstances, may result from either. But taking the table as it stands, periostitis is undoubtedly by far the most frequent form of osseous lesion.

Case X.—The following case, the report of which I also owe to the courtesy of Dr. D. S. Rice, of Hastings, Pa., who has had an unusually rich experience in the surgical results of typhoid fever, is a good illustration of typhoid osteomyelitis:

"Grace D., German, age fifteen, when twelve years of age had a severe attack of typhoid, lasting five weeks. She then convalesced rapidly, when suddenly, in the third week of convalescence, she complained of exceedingly severe pain in the middle third of the left tibia, without swelling or rise of temperature. A week after the pain began the temperature quickly rose to 105°, but the leg was only slightly swollen and was not discolored. An incision six inches long was immediately made, and the bone was trephined, evacuating three ounces of dark pus. The medullary cavity of the bone was then chiseled open for six inches, irrigated, and packed with gauze. The patient made an uninterrupted recovery, and has been well ever since."

In the ribs especially, I suspect that many of the cases classed as periostitis were really instances of osteomyelitis. Necrosis is most frequent in the tibia,

but in the ribs, as Paget[1] has pointed out, necrosis does not occur. On the contrary, it may occur, though rarely, in the costal cartilages. Helferich[2] and Berg[3] have especially called attention to the age and location in typhoid ailments of the ribs and costal cartilages. Periostitis, osteomyelitis, and chondritis are the usual forms of disease in the ribs and their cartilages. As the chest wall is thin, operations here are apt to be less thoroughly done than elsewhere on account of the danger to the pleura, the lungs, or the pericardium, or even of opening the pleura or pericardium, and hence these cases are peculiarly apt to be chronic and to require repeated operations. Stephen Paget[4] has related a case of periostitis of both tibiæ and the lower left costal cartilages, which only healed in the latter place after four or five operations in two and a half years. The following cases are good illustrations, both of the character and chronicity of the bone lesions of typhoid, in one of which the pleural cavity was widely opened without collapse of the lung or other disaster:

Case XI.—J. K., age forty-two, of Patton, Cambria Co., Pa., was kindly sent to me at the Jefferson Medical College Hospital by Dr. D. S. Rice, of Hastings, Pa., February 19, 1895. His father died of some gastric trouble; his mother of dropsy. Three sisters are living, all fairly healthy. No family history of tuberculosis or cancer. He was in good health until eighteen years ago, when he had an attack of typhoid fever which confined him to bed for ten weeks.

[1] St. Bart. Hosp. Rep., 1876.

[2] Berlin. klin. Wochen., Oct. 20, 1890, 979.

[3] Centralbl. f. Chir., 1896, 153. [4] Surgery of the Chest, 137.

Toward the end of this period he had some pain in the right sixth and seventh costal cartilages. When he got out of bed he discovered a small hard mass at the junction of these cartilages and their ribs. This gradually increased in size and was finally laid open, but no pus escaped. The wound closed, but the mass still grew larger and more tender, but without discoloration. In July, 1894, Dr. Rice incised the tumor and removed part of one rib and a portion of the sternum. The operation resulted in a small sinus, which never closed. A second operation was done in October, 1894. In January, 1895, the wound meantime never having healed, he was operated on again at the Pennsylvania Hospital by Dr. Thomas George Morton, who also removed a portion of the sternum and the parts of some ribs, but the wound never healed.

Status Præssens.—There are two sinuses, the upper one directly over the sternum and on a line with the nipple; the second about two inches below, at the right border of the sternum. Three inches from the nipple is a point of exquisite tenderness; all the tissues are indurated and thickened. His general health is good; he has neither cough nor night-sweats and has gained fifteen pounds in weight. There is no history of tuberculosis.

Operation, February 20, 1895. An incision four inches long was made over the sternum down to the bone. Two horizontal incisions were then made from the ends of the first to the line of the right axilla. The quadrangular flap thus made was dissected away, disclosing the sternum and ribs. The fifth, sixth, and seventh ribs were all found softened with osteomyelitis. Between three and four inches of each rib were removed as well as a large part of the sternum. In removing the fifth rib, in spite of the utmost care in loosening the tissues on the internal surface, a rent was made in the pleura in the

fifth intercostal space. This was closed by a finger while further dissection was being carried on, and later was stuffed with some iodoform gauze. In spite of this, however, the air was sucked in and out during respiration to a considerable amount. When the ribs had been finally removed, the gauze was removed and the opening inspected. It had then gradually been torn to an opening nearly 4 cm. in diameter. Through this with each inspiration the lung made a moderate hernia, and fell away again from the chest wall on expiration; no collapse of the lung occurred. During expiration I attempted to draw the edges of the pleura together with a needle (the pleura was entirely healthy), but it was so firmly attached to the margins of the opening that the attempt to tie the stitches simply made the hole larger by an additional tear in the pleura. Accordingly, I again stuffed the opening full of iodoform gauze and replaced the flap over it, allowing the end of the gauze to protrude at its lower border. His temperature on the day after the operation rose to 102°, fell by the next day to 101°, beyond which it never went, but reached the normal on the 11th of March, nearly three weeks after the operation. There was considerable local pain, the result of traumatic pleurisy, at the point where the pleura was opened. A few days after the operation, however, he was able to be out of bed. He was discharged on April 7, 1895. The wound, which at first had entirely closed, had reopened at two points, and there was evidently still further disease of the bone. In fact, at the time of the operation I was quite doubtful whether I had removed all of the diseased ribs, but in view of the probable pleurisy and of the very extensive removal of bone which would be required, and the possibility of a traumatic pleuro-pneumonia, I thought it best at the time to restrict the operation as already described. Cultures were made at the

time of operation from the sternum, ribs, and pleura, but they failed to develop any growth.

On December 24, 1895, Dr. Rice reported that the patient's general health was very good. The ends of the ribs, where they had been excised, were solid, and he could stand firm pressure against the chest wall without pain. The only discharge then existing was over the sternum.

He returned to the hospital on March 23, 1896, when I found a sinus leading down to the sternum, with a tumor the size of a pigeon's egg at the border of the ribs on the right side. The chest wall, where I had excised the ribs, was quite firm. The ends of the excised ribs could be felt, but the tissue between was filled up with firm fibrous tissue, which resisted pressure quite well. No evidence of the old pleuro-pneumonia resulting from the first operation could be detected.

Operation, March 25, 1896. A rectangular flap was turned up, the middle of which was at the sinus leading down to the sternum. An incision was then carried from the earlier one along the border of the ribs on the right side into the tumor already described. A soft spot was found in the line of the nipple on the seventh rib. After chiseling into this, I found that the spongy tissue was entirely broken down, the bony trabeculæ gone, and the medullary cavity filled with granulation tissue, which tunneled under the skin from the soft spot mentioned all the way up to the sinus leading to the surface. This I had not observed when I was operating on the diseased sternum. With the double rongeur forceps I removed the whole of the anterior wall of this tunnel, scraped out the medullary contents with a sharp spoon, and then used the Paquelin cautery to destroy any possible infection which had escaped the curette. His recovery was steady, but very slow. He went home, however, June 19, 1896, nearly three

months after the operation. Even then there was still a small superficial ulcer, from which, however, no sinus could be detected leading down to the bone.

Inoculations from the diseased bones were made by Dr. Kyle, and only staphylococci were found to be present.

In December, 1897, Dr. Rice stated that the patient is entirely well, and doing light manual labor.

Case XII.—I am indebted to Dr. Rice for the following additional case of presumed osteomyelitis of the ribs after typhoid fever: "A man, age forty, had a severe attack of typhoid fever in 1894, from which he made a good recovery. Three months after the attack a large inflammatory area formed over the sternum and ribs, and the ribs on the right side became so softened that they bent under very little pressure, and were so flexible that he was obliged to wear a bandage in order to ease his breathing. A small effusion of clear serum formed at the junction of the eighth and ninth ribs with their costal cartilages. This was drained aseptically, and under treatment with the phosphates the ribs became quite solid. But at the middle of the sternum, and at two points in the right chest and one on the left, the disease has recurred in December, 1897."

Case XIII.—I am indebted to Dr. D. S. Rice also for the following case of osteomyelitis of the left tibia: "A girl, age thirteen years, suffered from an attack of typhoid fever for thirty days. Two weeks after entire recovery she was seized with severe pain over the left tibia, four inches above the ankle. The temperature rose to 105° and the pain was so severe that six days after the onset operation was advised. The leg was but little swollen and there was no discoloration. The shaft of the tibia was exposed by an incision eight inches long and trephined. A large quantity of dark pus

escaped from the medullary cavity. The bone was chiseled out for a distance of six inches. She made an excellent recovery and has remained well for over a year."

Case XIV.—H. M. age eighteen, was kindly sent to me at the Jefferson Hospital March 19, 1896, by Dr. Hollenbeck, of Shamokin, Pa. At twelve years of age he suffered from a severe attack of typhoid fever, which lasted for ten weeks. While convalescing from the fever two weeks after defervescence, the inner surface of the left thigh just above the knee became swollen and slightly tender. In April, 1895, the swelling was lanced, and in October an operation was performed under an anesthetic. Eight months after the attack of fever the right ankle enlarged and suppurated. It ruptured spontaneously a year and a half ago, when fragments of bone were discharged and the abscess healed. The disease never interfered with his walking. The sinus above the knee has discharged continuously since the operation of last October, and is still open.

Operation, March 31, 1896. An incision was made from above the inner condyle of the left femur 12.5 cm. long, and the sinus traced from the inner border of the shaft of the femur some distance up the shaft, and also across the posterior surface of the shaft in the upper portion of the popliteal space. At this point a sequestrum was found 32 mm. long, triangular in shape, the base being 12 mm. in breadth. The femoral artery was exposed at the upper portion of the wound. The granulation tissue of the sinus was given to Dr. Kyle to examine for typhoid bacilli, who reported as follows: "No typhoid bacilli were found, but a pure culture of staphylococcus pyogenes aureus was obtained."

The bones seemed to have healed after the separation of the sequestrum, as no bare bone was discovered. All the granulation tissue was thor-

oughly removed and the wound packed with iodoform gauze. His temperature only once rose to 99.2°. The wound, however, like all typhoid wounds, was very sluggish in healing. He went home May 21, 1896, after two months in the hospital, the wound even then not being quite closed.

Necrosis of a lamella of bone lining the wall of the medullary cavity—*e. g.*, of the tibia, or what in my Toner Lecture I called "central necrosis," and of which I then collected three cases—does not seem to have been observed since 1876, except by Klemm,

Fig. 1.—Two foci of osteo-periostitis (*A* and *B*) following typhoid fever—"Shirt-stud Abscess" (*bouton de chemise*).

in a man of thirty-nine, whose left femur showed a sequestrum, 2 × 1 cm., lying in the pus. Very possibly this may be due to faulty observation, for I suspect it occurs, though only very occasionally. Colzi[2] has observed it also in one experimental case after five weeks of suppuration.

A not infrequent form of bone abscess which, if not understood, may be very imperfectly and incompletely treated, is that to which Chantemesse

[1] Arch. f. klin. Chir., 1893, xlvi, 885.
[2] Jordan, Beiträge klin. Chir., 1893, x.

has given the happy description "*bouton de chemise*," or "shirt-stud abscess." Cornil and Péan[1] give a very good illustration of this form of abscess, which, with their kind permission, I have reproduced (Fig. 1). In this form there is a localized abscess outside the tibia, probably under the periosteum, and another similar one under the outer layer of the bone or even in the medullary cavity, the two abscesses being connected by a cloaca or sinus through the wall of the bone—the shank of the "button." To treat only the external abscess will be fruitless. The bone must be trephined or chiseled into and the second abscess thoroughly evacuated, and its walls chiseled away if we wish a cure.

Sex.—As in the other surgical typhoid disorders, males largely preponderate, counting, in the two series:

Males,	123
Females,	63
Total,	186

Age.—Park[2] is in error when he says that two-thirds of the osseous lesions of typhoid occur in children and adolescents. This is true of even a much greater percentage in the joints (p. 102), but my tables show that in the bones there were under twenty, 56; from twenty to thirty, 71; from thirty to forty, 23; over forty, 16. Total, 166.

Taking thirty, however, as a dividing line, there were 127 cases thirty years of age and under, and 39 cases over thirty.

[1] Bull. Acad. de Méd., 1891, xxv, 602. [2] Mütter Lectures, 260.

So far as I have observed, there is only one part of the body in which osseous disease occurs almost uniformly later in life—the ribs and costal cartilages. The table shows that not a single case of disease of the ribs or their cartilages occurred before twenty. Of 32 cases, the age is given in 18: 5 were from twenty to thirty, 6 from thirty to forty, 7 over forty; that is, there were 13 over thirty—7 of whom were even over forty—to 5 under thirty.

Helferich,[1] who has observed eight cases, has called attention to the fact of the late appearance of disease in the ribs. Most of these probably should be added to the cases over thirty (for as the age is not given they could not be tabulated), and this would markedly increase the disproportion. He attributes it to the changes which occur, at least in the cartilages, in later life, an explanation which seems reasonable. It is noteworthy, also, that when disease attacks the ribs it quite as frequently attacks two or three or even four as a single rib.

Paget[2] twenty years ago noted, as has also Helferich, that, in sharp contradistinction to tubercular disease of the ribs, typhoid periostitis and osteomyelitis do not depress the general health. Indeed, in most of the bone cases this is true. The disease seems to be almost purely local. This naturally raises the question whether this may not be due to an acquired "immunity" of the system at large as a consequence of the fever. This seems at least a probable explanation of the undoubted fact that all of the lesions which occur during or after convalescence from typhoid cause the patients to suffer almost

[1] Berlin. klin. Wochen., 1890, 979. [2] St. Bart. Hosp. Rep., 1876.

wholly locally and to manifest but little febrile reaction.

Locally.—I have gone over the cases in the table and tabulated the various regions involved. Where more than one bone was attacked each bone has been entered upon the list and I find the results as follows:

Skull	[illegible]	
Mastoid	[illegible]	
Frontal	[illegible]	
Parietal	[illegible]	
Superior maxillary	[illegible]	
Inferior maxillary	[illegible]	
Head, total		24
Sternum	[illegible]	
Ribs	[illegible]	
Vertebrae	[illegible]	
Trunk, total		[illegible]
Clavicle	[illegible]	
Scapula	[illegible]	
Humerus	[illegible]	
Forearm	[illegible]	
Radius	[illegible]	
Ulna	[illegible]	
Hands	[illegible]	
Upper extremity, total		[illegible]
Pelvis	[illegible]	
Femur	[illegible]	
Tibia	[illegible]	
Fibula	[illegible]	
Foot	[illegible]	
Lower extremity, total		[illegible]
Total number of bones attacked		[illegible]

The first thing that strikes [illegible] distribution of disease of the bones [illegible]

parts of the body is, as is noted in the chapters on the Joints, Gangrene, etc., the extraordinary frequency of bone disease in the lower extremities,—112 as against 104 in all other parts of the body,—this, also, in spite of the fact that the part of the tibia most often involved—the antero-internal part of the shaft —has no muscles attached to it. This may be partly accounted for by slight accidental injuries, a possibility which can hardly be invoked in case of the femur except at the condyles. But as insisted upon in the chapter on Gangrene, I believe it is to be ascribed to the legs, *as legs; i. e.*, as the most distant part of the peripheral circulation, where nutrition is most sluggish and its activities most easily disturbed and impaired. That, as Parsons[1] has remarked, the foot, as well as the hand, is less often involved than most other parts of the body is to be ascribed, I think, largely to the more active circulation in bones, many of them at least made up largely of spongy tissue.

Taking the superficial bones—those of the head, sternum, clavicle, ulna, tibia, ribs, hands, and feet —as contrasted with the deep,—the vertebræ, scapula, humerus, radius, pelvis, femurs,—the relative number is as 180 to 51. Whether this has any significance in relation to the possible influence of accident is a question.

Date of Onset.—The facts here are both significant and instructive.

Of 186 cases, there arose:

In the first two weeks, ...	16
From the third to the sixth week, ...	66
From months to years after the fever, . . .	104

[1] He only found one case in the feet.

Bone disease is, therefore, rarely a complication of typhoid, but is, in the vast majority of cases, a sequel. It rarely arises during the course of the fever (the earliest case[1] arose on the tenth day), but almost always in convalescence, and, in fact, in over one-half the cases after convalescence was well established. It is at this time that muscular strain begins by the patient's commencing to stand and walk, and later by his return to ordinary manual labor; it is then that blows and other usual slight accidents may occur; it is then that the slow changes in the bones begin to show themselves; it is then that the bacilli, driven, so to speak, from other parts of the system, take refuge in the bones, and lie in wait, ready to avail themselves of any unfavorable conditions of fluctuating health or varying circulation, or of anything causing a *locus minoris resistentiæ*.

The experiments of Dmochowski and Janowski[2] are exactly in point. If the bacilli were injected under the sound skin of an animal no suppuration occurred, but if the tissue had been weakened by inflammation, by scars, or by local bleedings, then suppuration followed, or in some cases the animals died before any local changes could manifest themselves. In all pathologically-altered tissues suppurative action was apt to follow.

The inferences from these facts are plain. The subsidence of the fever, and even the establishment of full convalescence, do not justify the physician in relaxing his vigilance. No patient recovered from typhoid fever should be allowed to resume the ordinary activities of life except very gradually, and

[1] Ebermeier, Deutsch. Arch. f. klin. Med., 1889, xliv, 140. [2] *Loc. cit.*

should be cautioned as to even slight accidents and warned against any severe physical exertion for several months after he has apparently recovered. At the very first sign of trouble he should return to his physician for careful examination and advice.

Symptoms.—These will vary somewhat according to the type of disease, but only within rather narrow limits. The constitutional symptoms are rarely very pronounced, and fever especially is most commonly absent. In fact, so pronounced is this in the true typical typhoid cases, as distinguished from those arising from a pyogenic or a mixed infection, that Chantemesse and Widal[1] call this type of abscess "cold abscess," though it has, of course, no relation to the tubercular abscesses commonly described by this name. A possible explanation of this peculiarity of post-typhoid lesions has been advanced on the preceding page.

The cases may all be classed as either acute or chronic. Even in the acute cases, fever, anorexia, dry tongue, and constipation may be absent, the local symptoms predominating. Occasionally, however, in an otherwise chronic case, a temporary exacerbation may be attended with moderate constitutional symptoms.

Usually the first symptoms will be local pain, tenderness, and swelling, the latter being much more extensive than the bone disease which is its cause. In not a few cases resolution will follow after a relatively slow subsidence of the disease. But if, in a few days, or more likely a week or two, recovery

[1] Bull. et Mém. Soc. Méd. des Hôp., 1893, x, 779.

does not follow, the parts will become red and then, or soon after, may fluctuate.

In other cases, after an apparent subsidence of the disease, and even after weeks and occasionally months of supposed health, the pain and swelling will reappear at the same spot, and then go on to an abscess. Sometimes more than one such oscillation or recrudescence will take place, finally resulting either in suppuration or a permanent cure.

For some excellent examples of such oscillating cases see Osler and Parsons.[1] Routier and Terrillon [2] record a case in which the tibia was attacked in the ninth week during convalescence; the attack subsided, but was followed after some months by a relapse and an abscess, which was opened and the bone scraped. Two subsequent relapses and operations were required to effect a cure. Chantemesse and Widal [3] report a case illustrating both the multiplicity and the recrudescence of the bone lesions. Lockwood [4] reports a similar case of disappearance and reappearance of the swelling in an abscess. On pages 20 and 118 I have given some other instances of a similar character illustrating this curious tendency to the swinging of the pendulum between apparent health and renewed disease, and the case of H. W. (p. 123) is not dissimilar.

Treatment.—Fortunately, surgical treatment in most cases is needed, not during the typhoid attack, but after return to health. When fluctuation can be perceived, unquestionably immediate operation should

[1] Johns Hopkins Hosp. Reports, vol. v. [2] Prog. Méd., April 12, 1884.
[3] Bull. Soc. Méd. des Hôp., 1893, 3d series, x. 779.
[4] Lancet, 1895, i, 535.

be done. It is better, however, to operate even before fluctuation arises unless spontaneous resolution is fairly certain to follow. By such early interference suppuration may be avoided and the case cut short. If pus is found, then the cavity must be thoroughly treated. The bacilli are more often found in the tissues forming the wall of the abscess cavity than in the pus. Hence, if the abscess exists only in the periosteum and soft parts, these must be entirely excised and the bone be chiseled away. If, as not seldom happens, the medullary cavity is involved, then the bone must be widely chiseled away so as to expose and remove all the diseased osseous tissue. For want of thoroughness, repeated operations may be needed, the earlier attempts not having entirely removed the disease. Sometimes, although no pus is found, extensive operations are required before definite healing takes place. Thus, Chantemesse[1] relates a case in which for osteomyelitis the tibia was three times trephined. No pus was found, but the disease persisted, and the patient was only cured, a year later, by opening the tibia by an extensive operation forming a long gutter in the bone. Even then no pus was found.

In no region are these remarks more true than as to the ribs and sternum. From their very situation and their important relations to the pleura, pericardium, and lungs, incomplete, and therefore insufficient operations are apt to be done, and consequently have to be repeated. As a rule, the ribs must be resected in their entire thickness, and not merely partly gouged away. Sometimes it may be

[1] Bull. et Mém. Soc. Méd. des Hôp., 3d series, vii, 1890, 655.

difficult to say just where the disease stops (*cf.* Case XI, p. 129). The same rule applies to the sternum. If the pleural cavity is widely opened, as in my case just referred to, little or no mischief may be done, even in a suppurative case, provided the surgeon uses rigid antisepsis.

The form of abscess already described as "*bouton de chemise*," or as I would prefer as perhaps more accurate, "dumb-bell" in shape, must not be forgotten, and the inner as well as the outer abscess be treated as above directed.

The occurrence of this form of abscess again suggests the probably more frequent occurrence of "central necrosis" than is mentioned in the recorded cases. The sequestrum being small and bone chips from the chiseling being present may account for its not having been observed. Only those who know of its possibility would probably be on the lookout for it. Probably Parsons' Case III was an example in point.

Incomplete operations or neglected disease lead to sinuses, which may persist for years. In Sultan's case this had extended to six years, and yet no pyogenic infection of the sinus had occurred, a pure culture of the typhoid bacilli being found.

In Anger's case[1] the sinus continued for ten years, and only healed after the removal of a sequestrum.

The tendency to chronicity, to persistent sinuses, and especially to recurrences, are among the most marked characteristics of the bone disorders following typhoid. Occasionally the disease of the bone may

[1] Turgis, Contrib. à l'Étude de l'Ostéopériostite Conseq. dà la Fièvre Typhoïde, Thèse de Paris, 1884.

cause extensive disease in the soft parts; or may extend to a neighboring joint, though either complication is rare. Amputation in the latter case becomes imperative. Only four such amputations occurred in the cases reported in the first series; two died, one recovered, and one was under treatment. In the second series no case came to amputation.

The *prognosis* is almost always favorable. Of the 168 cases in the second series, only 11 died; recovery followed in 122 cases, the result not being given in 35. Of all the 11 fatal cases, not one can be said to be a pure case of typical bone disease from typhoid. Every one suggests either a pyogenic or a mixed infection. In only two were bacteriological examinations made. In Klemm's[1] case the colon bacillus, as well as that of typhoid, was present; the periosteum was destroyed and the bone-marrow red and hemorrhagic. Half a liter of fluid was found. In Dunin's[2] case there was also a parotid abscess, and a mixed infection with the staphylococcus aureus was found. In another,[3] a mixed infection certainly occurred, as the patient had also erysipelas. In three,[4] the jaws were attacked and the tissues of the cheek became gangrenous. In the first there was also suppurative parotitis. These were quite surely mixed infections. Three others were probably cases of septicemia,[5] combined, in the first,

[1] Arch. f. klin. Chir., xlviii, Heft 4.

[2] Deutsch. Arch. f. klin. Med., 1886, xxxix, 369.

[3] Armieux, Rev. Méd. de Toulouse, 1875, ix, 42.

[4] Franklin, Lancet, 1879, i, 553; Lawton, *Ibid*, p. 685; Alexander, Breslauer aerzt. Zeitschr, 1887, No. 23, 271.

[5] Mercier, Rev. Mens. de Méd. et de Chir., 1879, iii, 21; Rondu, quoted by Witzel; Robin, Gaz. Méd. de Paris, 1881, No. 4, 559.

with diffused violent phlegmonous periostitis of the left femur and 1½ liters of pus. The other two have already been referred to (p. 92) under diseases of the joints, as both bones and joints were involved. In a case of Furbinger's,[1] there was hemato-pyo-pneumothorax from perforation of a necrotic pulmonary focus. In the eleventh case,[2] the patient died from erosion of the popliteal vessels after operation on a femoral periostitis which had persisted for four years.

These complicated cases show that in purely typhoid cases a fatal issue can be practically excluded. Recovery is assured, but is apt to be slow.

[1] Verhandl. Neunte Kongress inner. Med., 1890, p. 207.
[2] Terrillon, Prog. Méd., 1884, 285.

CHAPTER VI.

TYPHOID ABSCESSES.

One of the most frequent accompaniments of typhoid fever are small abscesses in the skin, or furuncles. So far as I know, their bacteriology has not been investigated, but the probability is that they would be found to contain only pyogenic bacteria.

Other abscesses arise from various causes. For instance, in the abdominal wall they may arise from muscular degeneration in the recti muscles (see Hematomata); or, again, they may arise by invasion from the intestine at a point of adhesion and possible perforation; *e. g.*, in Vance's[1] case, which arose in the seventh week, there was an abscess which opened at the umbilicus and was found to extend from the ribs to the ilium and posteriorly to the loin. Possibly they may arise independently of either, by the lodgment of the typhoid bacillus in the connective tissues, as in the case of Raymond.[2] In this case an abscess was found in the abdominal wall extending from the umbilicus to the mons Veneris, and the sole bacterium found was the bacillus of Eberth. In the loose connective tissue elsewhere we may have the same phenomenon. For example, in Harrison's[3] case, in which there were three relapses, a cellulitis of the neck developed two and a half months after the third relapse and in the seventh

[1] Med. and Surg. Reporter, 1893, 281.

[2] Pein, *loc. cit.*, p. 58.

[3] Lancet, 1891, i, 1207.

month after the beginning of the fever, and extended even to the apex of the lung, but did not go on to suppuration. Fraenzel[1] records a fatal case of mediastinitis in a man of fifty-two. Werner[2] also reports a case in which several sternal ulcers existing prior to the fever extended and perforated into the anterior mediastinum. The patient died from profuse hemorrhage, the cause of which was not ascertained even by a necropsy. Brieger[3] reports cases of abscesses in both axillæ, the post-anal region, and the thigh. In another case, reported by Daly,[4] an abscess developed to the left of the trachea an inch above the clavicle. In another[5] there were abscesses about the larynx and trachea, but the case was septic. In another, reported by Tuthill,[6] an abscess developed under the deltoid in which the Eberth bacillus was found in pure culture. In another, by Hanquet,[7] there was an abscess in the left axilla along with thrombosis of the right femoral vein, periostitis of the left femur, and necrosis of the right ulna. The following unreported cases have been seen by myself or by friends:

Case XV.—Mr. S., Haverford College, Pa., age thirty-four, has a scrofulous family history, with possible inherited syphilis, as indicated by necrosis of the turbinated bones. He is of a nervous temperament, an excessive smoker, habits good. He was first seen,

[1] Berlin. klin. Wochen., 1874, 97.
[2] Med. Correspondenzbl. Württemb. aerzt. Verein, 1859, xxix, 70
[3] Zeitschr. f. klin. Med., 1886, xi, 263.
[4] Med. and Surg. Reporter, 1882, 346.
[5] Robin, Gaz. Méd. de Paris, 1881, 559.
[6] Trans. Med. Soc. State of New York, 1895, 222.
[7] Arch. Méd. Belges, 1892, 305.

in consultation with Dr. G. E. Abbot, October 19, 1890. Without prior illness, and following severe mental strain and unusual physical exercise and exposure to wet, he was taken with three heavy chills, September 19, 1890. This ushered in a severe attack of typhoid fever. For five days his temperature ranged from 105° to 106°, though the pulse only once rose above 100. The maximum daily movements of the bowels was nine. There was no hemorrhage from the bowels, but he suffered three hemorrhages from the nose at the end of the second week. He was delirious for two nights, had considerable frontal headache, but neither temporal nor cervical pain. After the decline of the fever his recovery was rapid, and by October 15th he was out driving. On October 8th, the twentieth day after the beginning of the fever, he complained of soreness at the angle of the jaw on the left side. This was followed by moderate swelling and slight redness, which, however, were stationary. On October 16th, while driving, an accident occurred which caused him much excitement and fatigue but no physical injury. On the 17th the swelling had markedly extended. There was some trouble in swallowing and the voice was slightly hoarse. When I first saw him I found a very tense condition of the tissues of the neck from the ear to the level of the cricoid. There was only moderate tenderness, and at no point was it clear that an abscess was tending to point, though such an abscess, of course, was immediately suspected. His general condition was good, strength and appetite satisfactory, pain not severe, and though it lessened his sleep, it did not entirely prevent it. The progress of the case was slow. At the end of a week there was some edema below the angle of the jaw, the voice and deglutition were a little more affected, and there seemed to be every reason to suppose that sup-

puration was taking place, in spite of the fact that he had not had any more pain and there was no fluctuation.

Operation, October 26, 1890. On that morning a small amount of pus escaped through the external meatus. The abscess was reached at a depth of 4 cm. of tense edematous tissue under the deep fascia, and four ounces of pus were evacuated. The abscess skirted the larynx and pharynx. In a week he was entirely well. I regret very much to add that no bacteriological examination was made.

Case XVI.—Dr. D. S. Rice, of Hastings, Pa., sends me the notes of the following similar case of abscess of the neck:

"Mrs. R., age twenty-three, contracted a severe attack of typhoid fever in the early part of October, 1897. On the sixteenth day she had a severe hemorrhage from the bowels, when her temperature fell to 94°, but afterward rose to 105.5°. By the thirty-fifth day the temperature had fallen to the normal. A week later she had a chill and a rapidly developing deep abscess on the left side of the neck. In one week there was fluctuation. An incision evacuated half a pint of dark turbid pus. The abscess had extended under the trachea. She made a good recovery."

Occasionally these abscesses are multiple, as in Quaife's case,[1] in which they were situated over the tendon of the biceps, in the left thigh, over the tendon of the left peroneus tertius, over the left scapula, and in the substance of the left soleus. In Dunn's case[2] they arose at various points over the body, arms, and legs. In another,[3] there were abscesses

[1] Australas. Med. Gaz., Aug., 1885, 271.

[2] Univ. Med. Mag., Sept., 1895, 909.

[3] Laveran, Bull. Soc. Méd. des Hôp., 3d series, v, 90.

on the dorsum of the right hand, in the right arm, and the left fore-arm, with muscular degeneration in the recti abdominis, but no suppuration in the latter place. In the abscess only the staphylococcus aureus was found, although the typhoid bacilli were found in large numbers in the spleen.

In a number of cases, in such regions as the buttock and the thigh, it is often impossible to determine whether the abscesses arose from direct infection of the typhoid bacillus or from muscular degeneration followed by rupture and hemorrhages in the rectus abdominis; and whether they owed their origin to the pyogenic or to the typhoid micro-organisms was only occasionally determined by a bacteriological examination. In a few cases, however, we can reach definite conclusions on these points. Thus, an abscess in the muscles of the thigh [1] showed a pure culture of the Eberth bacillus. In a gluteal abscess,[2] also, a pure culture was obtained. The abscess in Hirsh's case [3] probably was secondary to arthritis of the hip-joint, which was followed by dislocation.

In Senter's case [4]—a case of abscess in the muscles of the thigh—thrombosis of the pulmonary artery caused sudden death. It may readily have been a direct result of the abscess.

There are three cases [5] of *perinephric abscess* recorded, but in none was a bacteriological examination made. As all arose during convalescence, it is

[1] Tinctine, Arch. Méd. Exp. et d'Anat. Pathol., 1894, vi, 1.

[2] Tinctine, *loc. cit.* [3] Annals of Surgery, 1896, 212.

[4] Des Abscès musc. dans. la Fièvre Typhoide, Thèse de Paris, 1880.

[5] Alrich, Univ. Med. Mag., Feb., 1890; Pearson, Brit. Med. Jour., 1891, i, 861; Adam, Australas. Med. Jour., 1887, ix, 182.

not improbable that they were due to a true typhoid infection.[1]

One *lumbar abscess*[2] seems clearly to have been connected with an old psoas abscess, and was

[1] The two following cases, one of a perinephric abscess originating in a renal abscess, and the other of renal abscess and meningitis, were published after the tables were completed. They are so important and interesting that I append résumés of both from the Bull. et Mém. Soc. Méd. des Hôp., 1897, No. 2. Their typhoid origin was conclusively shown by cultures.

Fernet and Papillon record the case of a man who entered the hospital on the sixth day of the fever. On the thirteenth day he complained of pain in the left flank at the level of the enlarged and tender spleen. By the twenty-first day the temperature had fallen nearly to the normal, yet his general condition was worse. In the lumbar region was a tumor, rounded, elastic, dull on percussion, in front of the spleen, yet reaching to the lumbar region. On the twenty-fifth day Tuffier operated, evacuating a liter of pus. The transverse colon had to be displaced to reach the tumor. By the thirty-second day peritonitis developed, and he died a week later. The necropsy showed that the abscess had begun in the renal tissue, had dilated the pelvis of the kidney, and, rupturing, had produced also a perinephric abscess. Other renal abscesses were present.

Cultures from the pus evacuated at the operation and from the pus of the renal abscesses showed pure cultures of the typhoid bacillus; cultures from the dressings some days after the operation and from the renal pelvis at the necropsy showed the colon bacillus and saprophytes. Evidently, there had been a prior intermittent hydronephrosis, as shown by a marked kink in the ureter. The typhoid infection readily seized upon such a weakened organ, and all the rest is clear.

[Not only is this case interesting from the pathological and clinical side, but it teaches the lesson that if any abnormal condition, such as movable kidney, exist, it should be remedied at once, in order not only to cure the existing malady, but to avoid later indirect dangers which may easily follow.]

Troisier and Sicard report a case of renal abscess and meningitis of typhoid origin as follows: A man entered the hospital about the fifteenth day without characteristic symptoms of typhoid. He was suffering from lead palsy and the nervous symptoms of saturnism, including headache. The urine was markedly albuminous. Five serum-tests responded positively. Four days before death symptoms of meningitis appeared. He died on the thirty-second day. The intestine showed typical typhoid lesions, the spleen was diffluent. At the base of the brain in a yellowish fibrino-purulent exudate, and, also, in an abscess of the kidney, the bacillus of Eberth was found in pure culture. No symptoms attributable to the renal abscess, other than the albuminuria, are mentioned.

[2] De Lannoy, Phila. Med. Times, July 15, 1882, 707.

probably only lighted up afresh by the typhoid attack.

The following case of typhoid during an absolutely latent Pott's disease is of the greatest interest, both from the perhaps excusable error in diagnosis, owing to the peculiarly misleading history and symptoms, and as illustrating the value of a bacteriological and microscopical examination, without which a finally correct diagnosis would not have been made:

Case XVII.—George W., age twenty-seven, entered the Jefferson Hospital August 27, 1897, under the care of Dr. J. A. Salinger, on the fifth day of an attack of typhoid fever. Typical *tâches rouges* appeared on the next day. By the sixth day the temperature was normal. A slight relapse, with a second crop of spots, occurred from the thirteenth to the seventeenth day. He left the hospital September 22d. He re-entered my surgical ward on the 1st of November. He stated that for two weeks after leaving he took moderate exercise, ate heartily, and felt very well. Early in October some sharp pains were felt in the right groin at intervals for a week, and then ceased. They seemed to follow short walks. The pain extended into the thigh, and was especially noticeable on rising from the sitting to the standing posture. On October 10th he first noticed a painless tumor in the right groin. On his re-admission, November 1st, there was a soft oval tumor, 6 by 4.5 cm., just above Poupart's ligament on the right side, and just to the right of the internal ring. The tumor was globular, and showed distinct impulse on coughing to touch and to sight. When he lay down it diminished and almost disappeared. It could be reduced, and then two fingers could be inserted into an oval aperture in the belly wall which exactly resembled a hernial ring. There was no discoloration of the

skin. Manipulation of the tumor was not painful. The temperature did not exceed 99°. My impression was that it was a hernia.

Looking back on it, I think I ought to have been put on the right track by considering: First, that no known case of hernia attributable to the fever is on record, so far as I know. Secondly, abscesses are quite common. Third, had it been a hernia, it was too soft for omentum and also lacked the doughiness of such a tumor. If bowel, it should have been tympanitic, but it was distinctly dull. When reduced, also, no gurgling occurred, but this I attributed to the apparently wide open ring. I think I should also have been put on my guard somewhat by the fact that it was not precisely in the position of the internal ring, and, in addition to that, one does not usually feel the internal ring as distinctly as this was felt.

My impression was that the hyaline degeneration of the muscles of the belly wall, so constantly seen in typhoid fever, had been followed by a rupture of the muscular fibers, and this had led to the hernia. If this were the origin of the hernia, I deemed it wise to wait until distinct regeneration of the muscular fibers had occurred, which would give greater assurance of a cure. After a week in bed, however, as the tumor was increasing in size, I thought best to proceed at once to operate.

[illegible] November 10, 18[illegible]. An incision parallel with Poupart's ligament very quickly opened an abscess. Not less than a pint and a half of pus was evacuated. The pus was not [illegible] nor did it have any fecal odor. A probe passed upward [illegible] [illegible] it also passed down[illegible] [illegible]

[illegible]

with a rubber tube and a small amount of iodoform gauze.

The scrapings and a culture from the pus were given to Prof. Coplin for bacteriological examination.

My impression now was that instead of a hernia we had to deal with a typhoid abscess arising in the connective tissue between the iliacus internus muscle and the iliac fascia. One other possibility occurred to me: that it might have arisen from a perforation of the appendix by a typhoid ulcer, and that this had been followed by an abscess with a quiet afebrile course, as one sees not uncommonly after typhoid fever. I asked Prof. Coplin, of course, to make a very careful test to determine the presence or absence of the typhoid bacillus. After four weeks no growth occurred in the culture. In the scrapings from the abscess cavity, tubercle bacilli but no other organisms were found. I received the preliminary report at the end of a week, and this at once suggested to me that it was possibly a case of Pott's disease, followed by a psoas abscess. In conjunction with Prof. H. Augustus Wilson, I examined the man's back with the greatest care. Up to that time he was not aware that he had ever had any trouble whatever with his back. He had never suffered any pain, and had it not been for the microscopical demonstration of the cause of the abscess, I would never have suspected that it arose from vertebral caries. A very careful examination, however, showed unquestionably that he had a Pott's disease at the dorso-lumbar junction, and I placed him under Prof. Wilson's care.

All of the symptoms were now perfectly explicable. From the surgical point of view they are of no little interest, as one might easily be deceived, as I was; and from the pathological side it is also of interest, as it showed that a man who has had Pott's disease for a period of time long enough to produce a psoas abscess containing a pint and a half of tubercu-

lar "pus," and to travel from the spine down to the level of Poupart's ligament, can pass through an attack of typhoid fever and a relapse without having any infection in the abscess or the bones from the typhoid bacillus; without aggravating the Pott's disease in the slightest degree; and, indeed, without the patient's ever knowing that he had any disease whatever in the spine.

In the *abdominal cavity* abscesses may arise from several causes, and in any particular case it may be often difficult and not seldom impossible to determine definitely the cause.

They may arise as localized abscesses from perforation when the adjacent coils of intestine become adherent by a protective peritonitis and thus form a localized abscess. Elsner[1] and many others report such cases. They resemble the familiar form of an appendicular abscess, in which the coils of intestine become agglutinated with a similar result. In fact, both the appendix, as in non-typhoid cases, and the cecum in typhoid, may be perforated when no such adhesions exist,[2] and thus require operative measures (see Chapter XV, on Perforation); or, in other cases, perforation and adhesions may only be suspected and can not be proved.

Thus, in one case,[3] after a protracted course and a severe relapse an "ileocecal abscess" formed, and the pus had a fecal odor. In another,[4] a man of thirty-eight is said to have had a "perityphlitis" on the

[1] Trans. Med. Soc. State of N. Y., 1892, 314.

[2] For perforation of the appendix, see Bontecou, Jour. Amer. Med. Assoc., 1888, i, 106; Alexandroff, quoted in N. Y. Med. Jour., 1894, ii, 341.

[3] Pearson, Brit. Med. Jour., 1891, i, 801.

[4] Adam, Australas. Med. Jour., 1887, ix, 182.

twenty-eighth day, and on the fiftieth to have passed two ounces of pus by the bowel. In a third,[1] "typhlitis" is said to have occurred. All three recovered.

The following is the only similar case I have seen:

Case XVIII.—Mrs. Mary B., age twenty-six, Bellewood, Pa., entered the Jefferson Medical College Hospital January 5, 1893. She had always been well until two years ago, when she had an attack of typhoid fever lasting for four or five weeks. In her early convalescence (the exact time is uncertain) an abscess about the size of a goose egg formed in the right side and opened into the ascending colon. She did not have very severe pain. She passed a considerable amount of very offensive green-colored pus in her stools. About two months later a second abscess formed lower down than the first. It was very painful, and was opened by her physician in October, 1892. Fecal matter then began to be discharged with the pus, and a fecal fistula still exists.

Operation, February 11, 1893. An incision was made parallel with Poupart's ligament, the center of the incision corresponding to the fistula. After a very troublesome dissection, the fistula was found to end in a somewhat globular mass under the abdominal wall. This mass was seven cm. in diameter; the exterior was smooth and covered with peritoneum on the surface next the abdomen. On the other side it was attached to the surrounding tissues. The interior was made up of granulation tissue and pultaceous matter. No opening could be detected leading into the bowel.

Unfortunately it did not occur to me at the moment to inflate the colon with hydrogen gas or air which would probably have shown the intestinal end of the fistula. I suspected that the appendix was imbedded in the mass; that during the fever per-

[1] [illegible] Pepper, [illegible] Med. Mag., [illegible]

foration of the appendix had taken place ; and that this was the source of the fecal fistula. After searching as long and widely as I deemed prudent, but unsuccessfully, to find either the appendix or the opening into the bowel, the mass was curetted, disinfected, and packed. The peritoneal cavity had been freely opened, but the intestines were protected by iodoform gauze and no ill result followed. The fistula slowly contracted after the operation, so that when she was discharged, April 22, 1893, often two or three days elapsed without any discharge. Her general health, however, was poor. She died from general debility nine weeks later.

Typhoid ulcers in the appendix or caput coli may easily have produced such conditions, and even if no perforation take place, it is not impossible that the colon bacillus may pass through the intestinal wall during an attack of typhoid and produce an abscess, as has been frequently demonstrated in other conditions than typhoid. I believe there is no case yet recorded of the typhoid bacillus having been found free in the peritoneal cavity except where its presence has been readily accounted for, not by penetration through the intestinal walls, but from such sources as a perforation (Chap. XV), the rupture of abscesses of the liver (Chap. XVI), spleen (Chap. XVII), of the gall-bladder (Chap. XVI), or rupture of a mesenteric gland. In fact, just as in general surgery peritonitis is now believed to arise only as a positive result of a direct injury or of some usually demonstrable lesion in an abdominal organ, so peritonitis during or after typhoid fever always has a similar positive, but not always a demonstrable, cause. In a number of other cases perforation has

been suspected as the cause of an abscess without its being possible to be sure of one's ground. Thus, in Major's case,[1] on the eighteenth day the symptoms of collapse occurred. Three weeks later an abscess burst through the rectum, and the patient recovered. The author believes that a perforation led to the abscess. In Low's case,[2] in the third week the symptoms of collapse and peritonitis led the author to diagnosticate a perforation. These symptoms gradually subsided, and finally a residual abscess burst through the abdominal wall, gave vent to horribly fetid pus, and this patient also recovered. While both very likely resulted from a perforation, recovery prevented demonstrative proof. They are good illustrations of the witty dictum that "no case is *complete* without a post-mortem."

This complete proof, unhappily, however, is not always wanting. Thus, in a case of Lehmann,[3] in the third week perforation occurred. The patient died at the end of a month, and the healed scar of the perforation and the resulting encapsulated abscess were both found. There was also a suppurative parotitis due to a mixed staphylococcus and typhoid infection. The pus of the abdominal abscess showed a pure culture of Eberth's bacillus. This fact is worthy of especial note, as the perforation presumably gave ready exit to the colon bacillus. In another case[4] a man of twenty-five, just recovered from scarlatina, suffered from a mild attack of typhoid. At the necropsy, only six Peyer's patches

[1] Brit. Med. Jour., 1891, i, 18. [2] Brit. Med. Jour., 1881, ii, 122.
[3] Centralbl. f. klin. Med., 1891, 649.
[4] Potain, Gaz. des Hôp., June 9, 1891, 621.

were found to be inflamed, but all extended to the peritoneal coat of the bowel. He perished from a perforation which, not having any prior protective inflammation, led to general peritonitis, as in so many cases of perforative appendicitis without adhesions. In Elsner's case,[1] in spite of such a protective peritonitis marching in front of perforation, the patient, a young man of twenty-seven, died from profuse hemorrhage.

Tüngel[2] relates a case in which a suppurating mesenteric gland near the cecum gave rise to an abscess which perforated a branch of the superior mesenteric artery and caused death.

Perforation of the rectum has already been mentioned (p. 159, Major's case). In this case a pelvic abscess burst into the rectum.

Case XIX.—The following case of ischio-rectal abscess following typhoid, perforating the rectum and resulting in a fistula, is kindly furnished me by Dr. I. C. Schureman, of Toms River, N. J.:

Miss X., age twenty, had a severe attack of typhoid fever for six weeks in the spring of 1897. In the third week she complained of intense pain in the region of the anus and sacrum. A few days later an abscess was discovered, and was opened half an inch to the left of and posterior to the anus, giving vent to a large quantity of very offensive pus. In the fourth week of the fever another point of fluctuation was discovered three inches above the anus near the sacrum. The pus was evacuated, and the two abscesses were found to communicate with each

[1] Trans. Med. Soc. State of N. Y., 1892, 314.

[2] Klin. Mittheil. aus dem Kaiserlich. Hamburg. allgemein. Krankenh., Hamburg, 1864; quoted by Langenbuch, Deutsche Chirurgie, Leif., 45 c. Erste Hälfte, 238.

other. The tissues over the left natis near the anus sloughed over an area 1½ inches in diameter, and were a long time in healing after convalescence was established. Her vitality was so low that nothing further than symptomatic treatment could be carried out. She was advised to enter a hospital after she entirely recovered, in order to have the abscess fully laid open and treated, but declined to do so. In July, 1897, during an absence from home, she was very ill for several weeks. Ten days after the illness began she passed pus from the rectum for three days, and has passed it at intervals ever since until a month ago (November, 1897), when it apparently ceased. During her convalescence she suffered a great deal of pain in the lower abdomen, but only a small amount of pain in the rectum.

But the results are not always so happy, especially when the perforation proceeds from within outward. In one case,[1] on the forty-first day, as a result it was supposed of cough, a perforation occurred on the anterior face of the rectum (sigmoid flexure ?), nine inches from the anus. The case resulted fatally. Another case[2] of perforation of the anterior wall was followed by further complications, but finally ended in recovery. The perforation of the rectum occurred between the twelfth and the twentieth days, and resulted in a pelvic stercoral abscess. Between the thirty-sixth and the forty-fourth days this abscess burst into the bladder. The feces, it is surprising to note, gave rise to no special trouble in the bladder. The case was operated on by the gorget and thermo-cautery. The opening in the bladder then healed spontaneously, and the patient recovered.

[1] Cockle, Lancet, 1882, i. 178.

[2] Debongnie, Arch. Méd. Belges, 1893, 225.

Still another case[1] shows the extensive ravages which may result from a perforation of the rectum. In a man of thirty-six, a posterior perforation of the rectum on the twenty-sixth day was followed by an abscess in the meso-rectum, and finally led to his death by erosion of a hemorrhoidal artery. (*Cf.* Tüngel's case above.) But this was far from all: the left leg was swollen and emphysematous all the way to the ankle. The post-mortem explained the reason. Pressure on the leg forced fluid and gas from the leg into the pelvis. The pelvis was full of feces, and the abscess had burrowed its way out of the pelvis between the pyriformis and the gemelli into the thigh after perforating the pelvic fascia. A pelvic abscess in a case of Bosnières[2] also burst through the vagina in a young woman of eighteen. Though she suffered also from necrosis of both tibiæ, she made a good recovery.

Another cause of such an "abdominal abscess" is a *suppurating mesenteric gland*. Of this, too, the post-mortem table has given us convincing proof. Thus, in a case of Lehmann,[3] an abscess due to such a gland was found, and the pus showed a pure culture of Eberth's bacillus. In Fraenkel's case[4] an abdominal abscess was due, presumably, to a mesenteric gland, four and a half months after the onset of the fever. This first attack had been followed by two relapses, by facial erysipelas, and in the seventh week by symptoms of perforation. The abdomen was opened and 1½ liters of pus evacuated, in

[1] Pryor, Buffalo Med. and Surg. Jour., 1881-82, xxi, 555.
[2] Thèse de Paris, 1890. [3] Centralbl. f. klin. Med., Aug., 1891, 649.
[4] Verhandl. Kongress inner. Med., 1887, 179.

which only the typhoid bacillus was found. The patient died later of ileus.

In at least one other case the proof has been attained by an abdominal section which, instead of following, prevented the patient's death. Michie,[1] by a timely section in the fourth week, evacuated and drained an abdominal abscess due to such a suppurating gland. Thomson,[2] by the aspirator, removed five drams of grumous matter, which the author attributed probably to a mesenteric gland. The case is obscured, not only by the mode of treatment,—which, however, a happy recovery fully justified,—but also by the fact that the patient was of a strumous predisposition, and eight days before the development of the disease (which was about the period of defervescence) had wrenched his back. Low's case[3] was very likely a similar one. In the fourth week after defervescence the temperature gradually rose; a lymphatic gland in the neck suppurated; a tumor as large as a cricket ball formed to the right of the umbilicus and discharged into the bowel.

The following case of *subdiaphragmatic abscess* is the only one I have found:

Case XX.—Klein[4] reports a case of left-sided subphrenic abscess, probably arising from the spleen, following typhoid fever, in which the typhoid bacillus was found in pure culture in the pus.

A man, age thirty-four, on June 24, 1895, fell ill

[1] Brit. Med. Jour., 1888, i, 1388.

[2] Glasgow Med. Jour., 1882, xvii, 244.

[3] Brit. Med. Jour., 1881, ii, 122.

[4] Ueber die Pyogene Wirkung des Eberthschen Bacillus bei Typhuskomplikationen, Inaugural Dissertation, Bonn, 1896.

after drinking some stagnant water. By the 3d of August he was free from his fever and left his bed on the 8th. On the 13th he again went to bed with a temperature of 103°, with increased dulness over the spleen, but without tenderness. From this date the dulness and a distinct tumor in the region of the spleen increased. On his admission into the hospital, under the care of Dr. Schmidt, on the 22d of August, the left lower half of the thorax was markedly rigid in the line of the nipple, with but little respiratory movement upon that side of the chest. The dulness posteriorly began at the lower border of the scapula. Nothing abnormal in the heart. The diagnosis was made of a subdiaphragmatic abscess, probably arising from the spleen. Aspiration on the 23d of August showed the presence of pus. On the 26th of August Dr. Sartoris operated. A portion of the seventh rib was removed in the axillary line. A puncture was made and pus was found. The puncture showed, however, that access to the suppuration was not direct, but that the pleura lay between. Accordingly, the costal pleura was opened without disclosing any pus. The diaphragmatic pleura was seen to be markedly arched forward and was in direct contact with the costal pleura, but without any adhesions. The two layers of the pleura were carefully sutured and an incision was then made into the abscess, which discharged a thinnish, clear brown pus without any fecal odor. A catheter was introduced, and by means of this a second incision for drainage was made above and to the left of the umbilicus. The amount of pus evacuated was three liters. The surgeon endeavored to recognize the spleen, but neither this nor any other viscus was recognizable. The wound was drained and the patient made an excellent recovery. The bacteriological examination seems to have been made with great care, and showed only typhoid bacilli, and the author adduces the case as an excel-

lent evidence of the pyogenic faculty of the typhoid bacillus.

Of the *symptoms* and *treatment* of these abscesses I need not say much, as they differ little if any from the symptoms and treatment of similar ordinary abscesses. Any operation during or immediately after so serious a fever as typhoid is to be deprecated unless it be unavoidable. The dangers of either alone may be great, but of both combined *must* be great. But, on the other hand, to allow a patient to die from an abscess, be it in the abdomen, the neck, the thigh, the buttock, or elsewhere, is still more to be deprecated. Unless, therefore, the associated conditions prohibit it, such abscesses should be treated, on general surgical principles, by antiseptic evacuation and, usually, drainage. The need for strict disinfection is especially evident, since if it be not carried out we may add a staphylococcus or, worse still, a streptococcus infection to an existing typhoid infection, and further and gravely imperil the patient's chances of recovery.

CHAPTER VII.

TYPHOID HEMATOMATA.

It is rather strange that while in the earlier table (see Toner Lecture, "Hematomata") I was able to collect so many cases of hematoma, in the second collection, even larger than the former, Dr. Westcott found only 17 cases. These add but little to the facts collected in the Toner Lecture. I must, therefore, refer the reader to that chapter, adding only a few data.

The abdominal wall was, as before, the principal seat of the degeneration. The recti were involved 13 times, the "abdominal wall" twice, the great pectoral and the superficial muscles of the neck each once. The last patient died of septicemia. All the cases followed typhoid.

The ages of these patients were much below those of the former series, as nine of them were from fifteen to twenty-five and six were over twenty-five, the oldest being thirty-nine. There were 13 males to three females, a larger proportion of males than in the earlier table. Two arose during the third, fourth, and fifth weeks, two two months and one in the fifteenth week after the fever, three weeks after a relapse. Three were only discovered at the post-mortem. Of seven treated properly by incision and drainage none died. Of six, in which no mention is made of an incision, all died.

The following case is the only one I have personally seen.

Case XXI.—Abscess of the abdominal wall and pelvis after typhoid fever.

D. C., age twenty-seven, was admitted to St. Agnes' Hospital June 29, 1891. On March 7, 1891, he had been taken sick with typhoid fever and was in bed until April 27th. On May 29th he again fell ill with what was pronounced a relapse. About June 20th he suffered considerable pain in the lower abdomen. This was soon followed by swelling, which increased quite rapidly. On admission, nine days afterward, a swelling 9 cm. long was found just above the pubes, apparently in the right rectus. It was quite tender. His temperature was 102°. Unfortunately, I did not examine it by the rectum, and therefore can not state the condition of the pelvis. As the skin was in a very bad condition from the constant application of iodin, I applied an antiseptic fomentation to the part, both to relieve his pain and to remove the shreds of skin, as there seemed to be no urgency in his case. July 4th I made an incision into the abscess, and found that it not only occupied the muscle, but also extended into the lower abdomen and pelvis, reaching to the bladder and the rectum. It had not, however, ruptured into the peritoneal cavity, but had pushed the peritoneum before it. Over a pint of pus was evacuated and the cavity was then drained with a rubber drainage-tube. In a little over twenty-four hours the temperature fell to normal, and in twelve days he went home.

It is gratifying that recovery followed in spite of the extensive mischief wrought before operation. This is partly due, probably, to the late period at which it arose. I have ventured to place it here

under the head of hematoma, though it may have been an abscess in the abdominal wall and not have originated in a hematoma properly so-called. Its position, however, strongly suggested hematoma. Unfortunately, no facilities existed at the hospital at that time for a bacteriological examination. The bacillus of Eberth, however, has been found in the pus of a similar abscess by Raymond and Strauss.[1]

[1] Pein, Thèse de Paris, 1891, 58.

CHAPTER VIII.

CEREBRAL COMPLICATIONS OF TYPHOID FEVER.

I HAVE already alluded to three cases of obstruction of the Sylvian artery (Gangrene, p. 77). All three were on the left side, and were probably thrombotic rather than embolic in origin. In one of them, a case of ambulant typhoid,[1] there was tubercular meningitis in a woman six and one-half months pregnant, and it may be that the Sylvian obstruction was not of typhoid origin at all. Osler[2] has published an excellent paper upon hemiplegia in typhoid fever. Haines[3] has also reported a case.

The other cases of cerebral complications are 19 in number, 4 of abscess and 15[4] of meningitis. One of the cases of abscess, an "abscess of the meninges" (see below), perhaps should be classed as a meningitis. In the 15 cases of meningitis, both the dura and the pia were attacked indifferently, and the exudate was sometimes serous, sometimes seropurulent, but in at least 6 of the cases was purulent. In two (Ohlmacher, *vide infra*) there were also 120 c.c. and two ounces of blood clot.

It is noteworthy that a bacteriological examination was made in all 15, and that without exception the bacillus of Eberth was found—in 12,

[1] Huguenin, Correspondenzbl. schweiz. Aerzte, 1879, 440.

[2] Jour. Nervous and Mental Diseases, May, 1890.

[3] Bull. Johns Hopkins Hosp., 1896. [4] Besides the case on p. 152, footnote.

it is expressly stated, "in pure culture." Wolff[1] has collected 174 cases of meningitis in which a bacteriological examination was made and bacteria were found in all but 1.15 per cent. In 2.87 per cent. the bacillus of Eberth was found. In one of Ohlmacher's cases a mixed streptococcus and typhoid infection was found. In two cases[2] the patients died so shortly after the cerebral symptoms began that, as the author of one[3] points out, there was not time enough for other bacteria to have set up the meningitis and then to have disappeared.

Two of the cases[4] are referred to on page 47 as asserted cases of typhoid infection without the characteristic typhoid lesions in the intestine.

In no one of the four cases of abscess was a bacteriological examination made, a most unfortunate omission. All of them occurred during or after 1884, and the bacillus was then well known. Of these four cases a résumé is given at the end of this chapter.

Two of the 3 Sylvian cases died and every one of the other 19. The dates of onset are significant. Of the whole 19 cases, 1 of the Sylvian and 5 of the meningeal cases arose in the third week; the other 2 Sylvian cases arose, one "in the course" and the other on the twenty-third day, two days beyond the third week. Three arose in the first week and one in the seventh week. Four of the meningeal cases were discovered at the post-mortem.

[1] Berlin. klin. Wochen., 1897, No. 10.

[2] Kamen, Internat. klin. Rundschau, 1890, Nos. 3 and 4; Fernet, Bull. Soc. Méd. des Hôp., 1891, 361.

[3] Kamen, *loc. cit.*

[4] Balp and Adenot.

Practically, therefore, of these 16 cases 7, and possibly more, arose in the third week, when the fever would be at its height.

Of the 4 cases of abscess, 1 arose "in the course" of the fever, and the 3 others on the fifty-sixth, fifty-seventh, and ninety-eighth days, two to three months after the fever.

Sex.—Of the 19 cases, 12 were males and 7 females.

Age:

Under twenty there were, ...	6 cases.
From twenty to thirty there were, . . .	8 "
Thirty-one, ...	1 case.
Thirty-three, ...	1 "
Forty-four, ...	1 "
Fifty-seven, ...	1 "
Soldier (an adult), ...	1 "
	19 cases.

The subjoined résumés of three cases of typhoid meningitis by Ohlmacher are the most recent and the most carefully investigated cases that have come under my notice. It will be observed that one was due to a mixed infection, but in two a pure culture of the typhoid bacilli was obtained. In one in which suppuration occurred a distinct endarteritis was found (Plate II). This fact is not only important in its bearing on the pathology of typhoid meningitis which has reached the suppurative stage, but it is possible that it may throw some additional light on the production of the form of gangrene alluded to on pages 63 and 70. It is true that in such gangrene there is no suppuration so far as we know, but it at least suggests the need for more accurate investiga-

tion to determine whether it is not possible that endarteritis may occur in the extremities, especially in the legs, and thus lead to gangrene.

Case XXII.[1]—Man, age twenty-five, died on the seventeenth day after admission, in the fourth week. At the necropsy typical typhoid ulcers corresponding to the fourth week were found in the ileum, with swollen mesenteric glands. When the head was opened he found meningitis and 120 c.c. of blood on the right side, with edema and a cloudy pia with yellowish flakes. The typhoid bacilli were found in the spleen, mesenteric glands, bronchial glands, and in the pia in the region of the hemorrhage.

Case XXIII.[2]—Man, age forty-six, died in the fourth week. The spleen was swollen to twice its normal size and was quite soft. In the brain was found a leptomeningitis, with 40 c.c. of turbid fluid in the distended lateral ventricles. The typhoid bacilli were found in the lung, the spleen, the mesenteric and bronchial glands, and the pia. "Countless myriads" were found, on microscopical section of the brain, in the pia, arachnoid, and in the meningeal

[1] Ohlmacher, Jour. Amer. Med. Assoc., Aug. 28, 1897, p. 410.

[2] Ohlmacher, *loc. cit.*

SUPPURATIVE TYPHOID LEPTOMENINGITIS. (Explanation of Plate II.)

Fig. 1.—Portions of two cerebral gyri with intervening sulcus and meningeal covering; meningeal membrane filled with inflammatory exudate. Small branch of middle cerebral artery seen in cross-section, showing endarteritis. Photographed with Leitz obj. 2, oc. 1.

Fig. 2.—Portion of arachnoid membrane from same section as Fig. 1, showing numerous typhoid bacilli. Drawn from a photograph with Leitz obj. 1/12, oc. 1.

Fig. 3.—From same series of sections as Fig. 1. Branch of middle cerebral artery showing acute endarteritis. Note the inner bounding membrane of the lumen and the numerous small cells between it and the intima. Some of the small cells are also seen within the endothelial lining. Photographed with Leitz obj. 7, oc. 4.

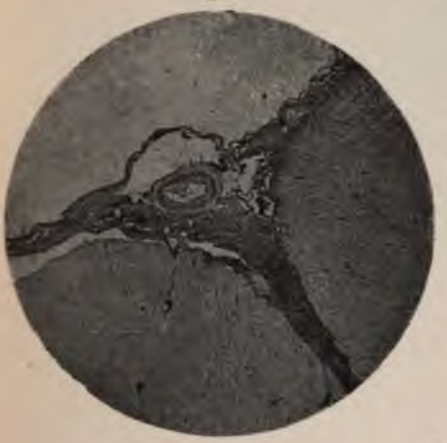
Fig. 4.—From another series of sections of the cerebrum in same case. Branch of middle cerebral artery showing complete obliteration of lumen by endarteritis. Same power as Fig. 3.

Fig. 5.—Same section as Fig. 1. Minute arteriole of middle cerebral artery, showing endarteritis. The endothelium is raised well into the lumen. There is moderate sub-endothelial cellular proliferation; a few cells lie within the lumen. Leitz obj. 1/12 oc. 4.

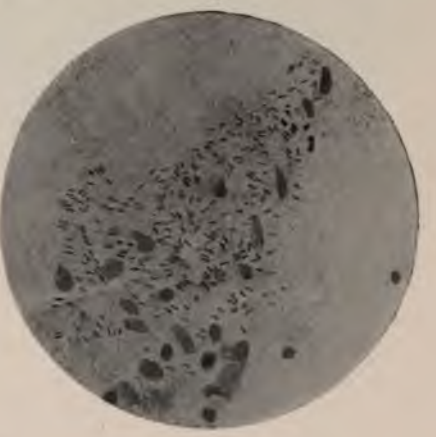
[Reproduced from A. P. Ohlmacher's paper (Jour. Amer. Med. Assoc., Aug. 28, 1897) by the kind permission of the author and editor.]

PLATE II.

1



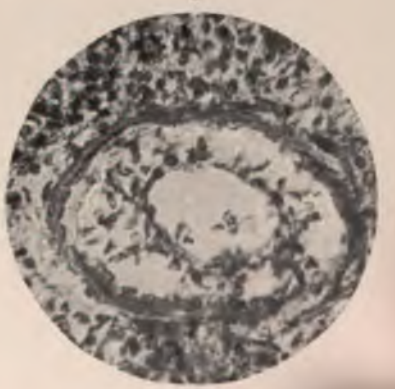
2



3

4

5



exudate. Extensive obliterating endarteritis was present.

Plate II is taken from Dr. Ohlmacher's paper, with his kind permission, and shows admirably the meningeal exudate, and especially the endarteritis, as seen in the preceding case.

Case XXIV.[1]—A woman, age twenty-four, who died at the end of the second week, having had tonic spasm of the flexor muscles, especially marked on the left side, preceding death. The necropsy showed three spots of ulceration in Peyer's patches. There was beginning left-sided broncho-pneumonia. On the right side of the brain there was acute pachymeningitis with about two ounces of clotted blood and a catarrhal leptomeningitis. In the blood from the heart were found cultures of the streptococcus. [It is surprising, in view of this, that there was no infective endocarditis.] In the spleen and mesenteric glands and in the blood of the heart he found pure typhoid bacilli. In the subdural space and in the pia he found the streptococcus and typhoid bacillus as a mixed infection. The streptococcus infection probably reached the brain by way of the lungs.

These three cases are particularly interesting by reason of the very careful microscopical and bacteriological examination which was made.

In the bacteriological examination a reference to his paper will show that he used all the more modern tests for differentiating the typhoid and the colon bacilli from each other, and the identification of the typhoid bacillus in the brain seems to be undoubted.

The microscopical examination is quite as interesting. In Case XXIII he found an "exquisite and

[1] Ohlmacher, Cleveland Med. Gaz., May, 1897, 409.

wide-spread acute endarteritis affecting the smaller arteries of the pia in both the anterior cerebral and the cerebellar regions; in other words, an endarteritis involving at least the branches of the middle cerebral and anterior cerebellar arteries. The process affects both the medium-sized and the smaller arterial branches, and varies from a partial to a complete obliteration. Serial sections from one of the larger vessels shows the advance from a partial to a complete obliterating endarteritis (Plate II). In the more minute twigs the change most frequently results in complete obliteration, and the newly-formed tissue fills the lumen of the vessel. The study thus far made of this condition seems to point to the conclusion that the endothelium of the intima is raised from its bed and forced into the lumen of the vessel by a sub-endothelial exudate; at least, a distinct membrane with fusiform nuclei can be found in the vessels in which the obliteration is not complete, bounding the new cells internally; and where the occlusion is complete a central core of cells, with laterally compressed nuclei, seems to be the same membrane forced into a compact mass. Two distinct kinds of small cells, both poor in protoplasm, are to be found in the endo-arterial exudate, one with nuclei composed of compact deeply staining chromatin, and engaged in one or another of the stages of indirect fragmentation; while the others, comparatively less numerous, are slightly larger and possess single nuclei with the chromatin more diffused. These small cells do not differ in structure from the pus cells of the general extravascular meningeal exudate, and are united by delicate protoplasmic fibers which seem to bind the cells together by a delicate reticulum, though they are often separated some distance from each other."

No bacilli were found in the arteries, though they were "abundant about the arteries, even to the ex-

tent of invading the perivascular lymph space, but they seem to be effectually barred by the muscular media."

The *symptoms* of cerebral involvement, especially of meningitis, are often overlooked. They are masked by the general stupor, and are supposed to be only connected with the usual delirium of typhoid. That only 15 cases of typhoid meningitis were found in such an extensive tabulation is remarkable. I am strongly disposed to think that such cases have been frequently overlooked by reason of the fact that the head is not very often opened in typhoid necropsies unless the cerebral symptoms have been very prominent. Even in cases without marked cerebral symptoms, examination of the brain, I believe, will show meningitis and infection by the bacilli of typhoid to be far more frequent than has been supposed heretofore.

Whether anything can be done by the surgeon to relieve such a complication is very doubtful, in my mind. Trephining would be useless, as the infection is too wide-spread. If I am right in this view, surgery has nothing to offer in such cases.

In **abscess of the brain** the symptoms are more pronounced, and may indicate both the nature of the lesion and occasionally even its location. When a hemiplegia or a monoplegia occurs, while it may be due to thrombosis, it should also raise the question of the possibility of abscess. If the eye symptoms point to this complication (optic neuritis), it would be confirmatory. But in cerebral abscess this is often wanting. The fever accompanying the disease its

would cause a rise of temperature, which generally is absent in ordinary cerebral abscess, and this will obscure the diagnosis. The same may be said of headache, which so constantly accompanies typhoid, especially of the cerebral type. Cerebral vomiting also would be ascertained with difficulty. On the whole, therefore, the diagnosis is difficult, and may be impossible.

Even if a fairly positive diagnosis can be made it is doubtful whether trephining would offer any loop-hole of escape from death. Yet, on the other hand, every such case so far has died, and at least no worse result could follow an attempt to relieve such a hopeless condition. Only of late have surgeons had the boldness to interfere in cases of biliary and intestinal perforation, and with at least fair results. Possibly the same may be true in the future, if we attempt to rescue also the cases of cerebral abscess. I should be inclined, at least, to try. Fortunately, as our tables show, such a complication is exceedingly rare.

I append a résumé of the cases of abscess of the brain, at least for our information and as food for reflection.

I also add an unusual case of *thrombosis of the intracranial veins* without involvement of the sinuses but with three small abscesses (?) of the brain.

Case XXV.—Hölscher[1] simply alludes to one fatal case of abscess of the frontal lobe of the brain in a patient aged twenty-four.

Case XXVI.—Josserand[2] reports a case of a young girl of twenty who died of suppurative menin-

[1] Münch. med. Wochen., 1891, No. 4. [2] Lyon Méd., 1894, vol. lxxvi, 97.

gitis in typhoid fever. She had moderate aphonia and right monoplegia; death followed forty-eight hours after the cerebral symptoms began. At the necropsy recent meningitis was found over the left lobe. It had its origin in an abscess as large as an orange at the base of the second frontal and the ascending frontal and parietal convolutions. It had depressed the cerebral substance without altering the convolutions, which were perfectly recognizable. The patient had a slight excoriation over the sacrum, only involving the epidermis and without suppuration. No bacteriological examination appears to have been made.

Case XXVII.—Deschamps[1] reports the case of a man, age nineteen, who entered the hospital December 4, 1893, with ordinary typhoid fever. On the 31st he had a relapse. By February he had entirely recovered, excepting for a persistent diarrhea. The temperature had become normal. On the 14th of February a pleurisy with effusion was observed. The patient began to grow weaker and showed signs of tuberculosis at the left apex. On the 28th of February he complained of severe pain in the right ear and the right face, with difficulty in deglutition, and facial neuralgia was diagnosticated. There was nothing abnormal in the throat. By the 22d of February his temperature had become normal. On March 1st, about the ninety-eighth day, it rose to 39.3° C. On the 2d, though it had fallen to 37.2°, a paresis of the right hand manifested itself. There was no paresis of the left arm. There was no disturbance of sensibility. The facial neuralgia persisted, and the patient for several days had bilious vomiting. He died on March 4th. The necropsy showed a serous pleurisy of the left side; tuberculosis of the left apex; a normal heart, liver, kidney, and spleen; congested, cicatrized, and thickened

[1] Progrès Méd., 1884, vol. xii, 950.

Peyer's patches in the intestine. There was no meningitis, but an abscess of the brain was found on the right side at the posterior portion of the first temporal convolution. It did not communicate with the ventricles in the left hemisphere; there was no lesion of the ear on the right side.

Case XXVIII.—Huchard and Tissier[1] report the case of a man, age fifty-seven, who suffered with all the classical symptoms of typhoid fever. He had a slight trace of albumin in the urine; his hebetude was very pronounced, but was more apparent than real on account of his typhoid deafness. He entered the hospital April 21, 1884, about twelve days after his illness began. He was obliged to remain in the hospital for six weeks on account of his slow convalescence, but without any complication that was perceptible until the 5th of June (fifty-seventh day) when, after an elevation of temperature, complete aphasia set in, with paralysis of the right side of the face. On the next day the arm was paralyzed; on the second day the leg of the right side was paralyzed; the paralysis in both being not quite complete. Sensation was diminished. The patient did not complain of his head, had not vomited, had no albuminuria, no valvular lesion. His belly was distended and the diarrhea had not ceased. On the 7th of June speech partially returned and his paresis diminished. On the contrary, his intellectual state became much worse. His torpor was excessive, so that he could scarcely be roused by any question. He died on the 11th of June. The necropsy showed that the heart was normal; the intestinal lesions of typhoid were marked. At the middle and upper portion of the left hemisphere a localized meningitis existed, which had resulted in an abscess of the meninges, 12 by 5 cm. The convolutions underneath the abscess were flattened out. The portion

[1] Progrès Méd., 1885, vol. xiii, 440.

of brain involved was the frontal and parietal lobes, especially the ascending frontal and parietal and the second and third frontal convolutions near the fissure of Rolando and the two parietal lobes. The contents of the abscess were between 50 and 60 gm. of green, creamy pus. There was no evidence of ecchymosis or infiltration. The abscess existed between the dura and the arachnoid. There was no suppuration in the petrous bone.

Case XXIX.—Dr. A. B. Richardson[1] relates an interesting case of marantic thrombosis of the intracranial veins following typhoid fever, without thrombosis of the sinuses. The patient was a man aged forty-three, who had had two injuries, one on the right side of his head twelve years before, the other on the left side of his head three months before, but without any cerebral symptoms except more or less headache. Toward the end of the third week defervescence took place. Just at that time, however, he began to complain of intense pain in the left temporal region, with, later, subnormal temperature and collapse, contraction of the pupils, somnolence, delirium, and convulsive movements in the left arm and leg and right face, and, later, on the right side of the body. Operation was considered, but decided against in view of the too diffuse and uncertain symptoms. The case was diagnosticated as probably one of acute meningitis. The eye grounds showed only general engorgement, more pronounced on the left side. He died at the end of about a week.

The necropsy showed no signs of meningitis, but the principal veins on the surface of the cortex were distended with hard clots; one of particularly large size lay along the fissure of Rolando. In the first temporal convolution of the left side was an area the size of a hickory-nut, which broke open, dis-

[1] Jour. Nerv. and Ment. Dis., July, 1897, p. 404.

charging a small amount of pus and broken-down brain tissue and blood coagula as the brain was removed. On the right side of the brain, just in front of the Rolandic fissure at its middle and upper third, was another area of a similar character though less advanced. In the right occipital lobe was another area, but not yet softened. The sinuses were entirely free. Unfortunately, no bacteriological examination or microscopical examination seems to have been made. Whether the collapse was the cause or the result of the thrombosis is uncertain.

CHAPTER IX.

OTITIS MEDIA IN TYPHOID FEVER.

Dr. Westcott has collected 31 cases, of which 24 are reported by a single observer.[1] Since the table was completed Hengst[2] has added 28 cases out of 1228 cases of typhoid collected from the practice of a large number of physicians—an average of about 2.5 per cent. Very likely many observed cases have not been deemed important enough to record. Only one case of mastoid disease was observed (Hengst) in all the 59 cases.

The usual cause is obviously a pyogenic infection from the throat. The diplococcus of pneumonia has also been found. In two bacteriological examinations[3] the bacillus of Eberth was not found, but its presence has been shown by Destrée and Vincent (p. 26). The infection, however, would be more likely to be pyogenic, access to the middle ear being obtained through the Eustachian tube. Cold draughts or cold water in the ear from bathing the patient may, as Hengst believes, occasionally cause the trouble, but I would hardly attribute much importance to them as compared with the infection from the air-passages.

The disease is a complication rather than a sequel.

[1] Sorel, Soc. Méd. des Hôp., 3d series, vi, 1889, 224.
[2] N. Y. Med. Jour., June 6, 1896.
[3] Dunin, Deutsch. Arch. f. klin. Med., 1886, xxxix, 369.

It arises usually in the second to the fourth week, when the patient is at his lowest ebb. The mouth and naso-pharynx are filled with mucus, which the patient is too weak or too indifferent to expectorate, and the same region is not seldom also the seat of ulcers which add pus to the mucus. Though, as Sorel points out, this complication usually accompanies mostly grave and prolonged cases, yet not a single death is recorded among the 59.

The symptoms and treatment call for no special remarks.

CHAPTER X.

TYPHOID PAROTITIS.

It is a noteworthy fact that in the later and larger series of cases Dr. Westcott only tabulated 50 cases of parotitis as against 378 in the Toner Lecture. I must, therefore, refer the reader to the Toner Lecture for the chief facts in relation to parotitis.

The difference in the numbers is easily explained when it is noticed that in the earlier series 362 followed typhus, a far more septic disorder than typhoid, and only 26 followed typhoid. In the present table all but one followed typhoid. The death-rate in the cases in which the result is stated is nearly 30 per cent., for of 28 cases, 8 died. Twenty of the 28 in which the sex is named were males. Of 25 cases the average age was nineteen years, an earlier age than the former series, and nine of them were children under fifteen. Suppuration was much more frequent in the present series, for of 34 cases, 29 suppurated and only 5 did not. In 12 cases the parotitis was bilateral, of which 7 suppurated on both sides.

The cause of the parotitis is undoubtedly generally a secondary infection by the pyogenic bacteria, which find many open ports of entry in the fissures of the lips, abrasions in the mouth, and a direct path by the duct of the parotid. In at least two cases, [1]

[1] Janowski, Centralbl. Bakteriol. und Parasitenk., 1895, xvii, 685; Lehmann, Centralbl. klin. Med., Aug., 1891, 649.

however, the bacillus of Eberth was found. In the last case it was associated with the staphylococcus, but in Janowski's case the typhoid bacillus existed alone. The patient was a man of twenty, who died in the second or third month of the fever. The suppurative faculty of the bacillus and its late ravages are thus once more demonstrated.

Doubtless, very many cases of parotitis have escaped notice in the histories, which would give the more salient points and omit a detail such as this. In very many cases, also, the titles have not named parotitis as a complication, and thus they have not been tabulated.

The *treatment* is the usual one for such complications—abortive, if possible by ice, iodin, ichthyol, etc.; but if suppuration occur, a free antiseptic incision and drainage.

The following case of continued *sweating in the parotid region* after a typhoid parotid abscess I owe to the courtesy of Prof. Osler. While not strictly surgical, it is sufficiently rare and curious to be made a matter of record.

Case XXX.—"B. F. A., age twenty-six, applied to the Johns Hopkins Hospital January 18, 1896, complaining of sweating over a limited area on the right side of the face and forehead. The patient had typhoid fever in September, 1890. During convalescence an abscess formed in the right parotid region, and was opened. Three or four months after the healing of the wound the patient noticed profuse sweating over the right side of the face and temple when eating, and he has been troubled with it ever since. When seen the patient was a healthy-looking, active man. There was a scar of the inci-

sion at the angle of the jaw two cm. in length. It was a little thickened and indurated, and slightly sensitive on pressure. Simple movement of the jaw, as in the act of mastication, produced no sweating. The chewing of food, more particularly on the right side, caused profuse sweating. The application of an electrical stimulus or of acid on the right side of the tongue caused it. The condition was the

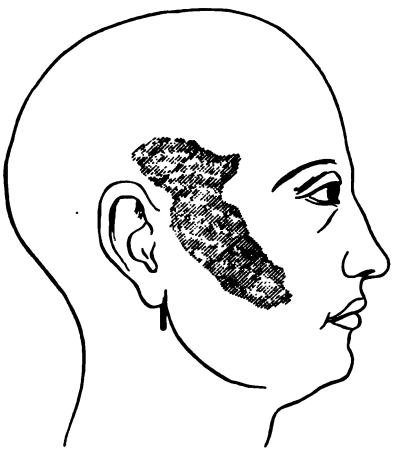


Fig. 2.—Area of Sweating in Case XXX.

source of a good deal of discomfort, as when eating his meals he had to mop the side of the face every few minutes. The area over which the sweating occurred (not strictly 'parotid' however) is shown in the diagram (Fig. 2). There was no facial paralysis and no disturbance of sensation.

"Of similar cases reported in the literature, a number of them have followed from the same cause; others after injuries in the parotid region."

CHAPTER XI.

TYPHOID AFFECTIONS OF THE THYROID GLAND.

THE presence of the typhoid bacillus in the thyroid gland has been affirmed by a number of authors (see p. 28). That the pyogenic bacteria should also be present would not be surprising; but in the ten tabulated cases in which the thyroid was involved, in only one[1] was the staphylococcus albus found, and then not alone, but mingled with Eberth's bacillus. In three others[2] the typhoid bacillus was found alone. In the last case, while there can be no question that the suppuration in the old goiter was due to the typhoid bacillus, it may possibly be a question whether the patient had really typhoid fever. The thyroid abscess began on the second day. No typhoid bacilli were found in the stools. If it was not a case of typhoid fever, then it would seem to be analogous to the cases referred to on page 43 of typhoid infection without the usual intestinal lesions of typhoid fever.

It may be merely accidental, but it is at least noteworthy, that of the ten cases in my table, in four suppuration occurred in thyroids already diseased—*i. e.*, in old goiters. This may have been a

[1] Spirig, Correspondenzbl. f. schweiz. Aerzte, Feb. 15, 1891.

[2] Colzi, Lo Sperimentale, 1891, No. 2; Dupraz, Arch. de Méd. Exp. et d'Anat. Pathol., Jan. 1, 1892, 76; Kummer and Tavel, Rev. de Chir., June, 1891, p. 507.

predisposing cause. The infection may readily reach the gland through the blood, even when this gland is normal; but when it is in the condition of cystic goiter, this is still more readily conceivable. Colzi, in his case, attributed it to hemorrhages into the cystic cavities caused by the constant cough, and that then infection of the blood clots easily occurred through the blood current. In all but one of the ten, suppuration occurred. In seven the attack began in convalescence—*i. e.*, after the third week; in two as late as the seventh or eighth week. Five were males as against two females. Two were "young"—presumably, therefore, under twenty; two between twenty and thirty; three were over thirty. Six recovered, and one, who had also an abscess of the spleen, died.

Several of Lüning's cases of the laryngeal complications of typhoid had also old goiters. They are tabulated in the next chapter and not here.

Treatment.—When suppuration has taken place, the only treatment, of course, is to open the abscess under careful aseptic precautions if the gland be normal; but if the case be one of old goiter, it may be better to extirpate the suppurating half, as was done by Kummer. This would only be done, of course, in convalescence. During the fever, only temporizing methods should be employed.

CHAPTER XII.

TYPHOID AFFECTIONS OF THE LARYNX.

THE larynx and the joints are the two sites of frequent surgical complications of typhoid upon the pathology of which the discovery of the bacillus of Eberth has had practically no influence. In Chapter IV I have related the results of bacteriological examination in typhoid arthritis. In not a single case has the bacillus been found in the joints, nor am I aware of a single observation which has revealed its presence in the larynx. Possibly such a case may exist, but if so it has not been found in the pains-taking search of Dr. Westcott.

The present chapter, therefore, will be little more than an enlargement in the way of statistics of the corresponding chapter in my Toner Lecture,—to which, in the Appendix, the reader must be referred, —together with the development of such additional facts as the larger number of cases will furnish.

But one attempt has been made since my lecture in 1876 to gather together the material needful for the consideration of the subject.[1] In 1876 I collected 169 cases. In 1884 Lüning, who had evidently not seen my lecture, collected 213 cases. Dr. Westcott has not collated the two series, as it would have been an unnecessary and tedious task. Presumably they are very nearly identical, excepting

[1] Lüning, Archiv f. klin. Chirurgie, 1884, xxx, 225.

the 14 original cases reported by Lüning and the 30 cases in excess of my first series. Dr. Westcott has, however, collected 38 cases since Lüning's paper, which, with Lüning's 14 original ones, added to the 169 of my first series, make 221. The following remarks are based, therefore, upon these 221 cases. I shall also quote, as occasion demands, some tables and remarks by Lüning, whose paper is not only very full and detailed, but of the greatest interest and importance both to the surgeon and the laryngologist.

Lüning's description of the symptoms is so graphic, and so useful in calling attention to the unexpectedness and the dangers of such cases, that I can not do better than to quote it in full (p. 286):

"The fact that the patient, at the time when the lesions in the larynx are so wide-spread that they ought to be recognized clinically, lies in typhoid apathy, and often makes no complaint of his suffering, is a great hindrance to the clinician. All the more is it his duty not to allow even slight complaints, especially if they arouse even a suspicion of any laryngeal complication, to pass unnoticed. . . . In the histories will be found the oft-recurring remarks, 'The patient was doing well,' or 'he was slightly hoarse,' 'complained slightly of dysphagia,' etc. . . . Physician and patient together rejoice over the daily progress toward convalescence; of the still slight but persistent trouble in the throat scarcely a word is said, until all at once—an exposure to cold, a little walk, is then usually blamed for it—the hoarseness increases, and swallowing becomes markedly painful. The picture now quickly alters. Soon, often within a few hours, come dyspnea and suffocative attacks. Sometimes even during the

very first day the anxious scene of laryngeal stenosis sets in, with stridor, inspiratory depression of the neck and chest wall—the unrest of despair, a struggle with death. The face becomes livid; the respiration becomes rapid, wearisome; the auxiliary muscles of respiration are all called into play; sometimes the respirations are prolonged and noisy. The patient can find no rest; the dyspnea even prevents the taking of nourishment; the expectoration of the increasing mucus becomes imperfect; soon attacks of suffocation recur. Either a tracheotomy must now be done immediately, or the patient, if he is weak, may choke to death, even in the first attack. More commonly, however, the attack subsides, and a slight improvement with a short sleep will ensue. Expectoration of bloody mucus, masses of pus, and, in some cases, even of pieces of cartilage, diminish the symptoms, and show at the same time that the real cause of the dyspnea is not a catarrhal edema or dropsical swelling but a destructive ulceration, even of the cartilages. Often, also, there is severe fever. Thus pass on, it may be even days and weeks, easy breathing alternating with the suffocative attacks. The alternative is only a finally fatal attack of suffocation or a late palliative tracheotomy with all its uncertainty. . . . If one will read the cases of death from suffocation without operation gathered in Table B (52 cases, 49 deaths), he will find that, almost without exception, suffocation occurred early and quickly, before either physician or patient had even thought of tracheotomy.

"This is the picture in cases of perichondritis. If the patient is in the stage of typhoid stupor, when the ulceration is accompanied with acute suppuration and swelling which may lead to destruction of the cartilages, the initial symptoms of the threatening danger may escape us entirely in spite of careful observation. . . . In these cases the objec-

tive signs of laryngeal stenosis, on which we usually depend, are much less marked; stridor, movements of the larynx, inspiratory depression, action of the auxiliary inspiratory muscles—in short, everything by which, in the healthy, we make the diagnosis of narrowing of the air-passages is, in the *vita minima* of the weakened patient, far less outspoken, and easily deceives us as to the degree of the danger of suffocation. The striking suffocative attacks, with arrest of respiration, so alarming even to the lay observer, are less noticeable, since the struggle of the patient with the mechanical obstruction quickly fails or is quickly abandoned. The condition passes into a death agony, with edema of the lungs, without the stenosis seeming to have reached a threatening degree. . . . And thus one sees, often with astonishment, in the reports of the necropsies, how often the stenosis and destruction of the cartilages occurs, as it were, 'without even any symptoms.'"

A vivid picture like this may well give both the physician and the surgeon food for thought. In America, however, as in England, it would seem that cases of perichondritis are less frequent than in Germany. The influence of nationality was not alluded to in my Toner Lecture, nor was the nationality of the cases in the second series tabulated. Lüning, however, has done so in his 199 cases from surgical literature, and found that of the 147 cases subjected to tracheotomy, 117 were from Germany (including Switzerland), 16 from France, and only 8 from England. Of the 52 not tracheotomized, 37 were French, 11 German, and 4 English.

Two other points not mentioned in my former paper are the occurrence of emphysema

extension of the suppuration to the mediastinum. Both are evidences of the gravity of the affection.

Thus, Wilks [1] records the case of a child of twelve who on the twelfth day developed general emphysema, due to a perforating ulcer on the posterior wall of the larynx. Durham [2] records a similar case in a boy of ten, and Chomel [3] a third in a man of twenty, from an ulcer which perforated the thyroid cartilage.

In one of Lüning's cases (IX) an abscess existed in which no trace of the arytenoids could be found and the cricoid was undergoing necrosis. The anterior mediastinum was infiltrated with pus. In another case [4] a similar abscess existed around the bare thyroid cartilage, and both the anterior and posterior mediastina were involved.

Following now the order of my Toner Lecture, to which the reader is referred for a fuller discussion of each point, I will give the revised statistics of the two tables combined.

The total number of cases collected is 221, of which at least 89, and probably many more, certainly involved the cartilages.

Age.—Of 146 cases there were:

Under fifteen, ...	12
Fifteen to twenty-five, ...	87
Over twenty-five, ...	47
	146

showing that childhood is remarkably exempt from laryngeal complications.

[1] Med. Times and Gaz., 1862, ii, 276.

[2] Holmes' Syst. Surgery, 2d ed., iv, 571. [3] Thèse de Paris, 1877.

[4] Retslay, Ueber Perichondritis Laryngea, Berlin. Diss., 1870, No. 10.

The corresponding table of Lüning, including his own 14 cases, gives:

Age.	Males.	Females.	Total.	
Under ten,	4	2	6	18
Ten to fifteen,	7	5	12	
Fifteen to twenty,	30	11	41	109
Twenty to twenty-five,	56	12	68	
Twenty-five to thirty,	25	3	28	38
Thirty to thirty-five,	3	2	5	
Over thirty-five,	4	1	5	
	129	36	165	

The frequency from fifteen to twenty-five (109 to 56 for all other ages) is doubtless explained by the greater prevalence of the fever at that age.

Sex.—Of 148 cases in my tables there were:

Males,	119
Females,	29
	148

and in Lüning's table, as above, there were:

Males,	129
Females,	36
	165

Site of the Stenosis.—This was:

Supraglottic,	50
Infraglottic,	36
In the glottis,	18
	104

This would seem to bear out the view of Lüning that the arytenoids are the most frequent site of the perichondritis at its beginning, and that from these the disease extends to the cricoid.

Date of Onset.—This is generally late and fre-

quently during convalescence, when the fears of the physician are apt to be lulled by returning health.

First week,	7 cases.
Second week,	23 "
Third week,	30 "
Fourth week to two months,	83 "
	143 cases.

Typhoid vs. Typhus Fever.—In my Toner Lecture a considerable number of cases following typhus fever (49) were tabulated; in the second series only four. Combining the two tables there were:

Following typhoid,	154
Following typhus,	53

Necrosis of the Cartilages.—This is by far the most common and also by far the most dangerous form of laryngeal affection, for of 75 cases 71 died—a mortality of almost 95 per cent. Of the four recoveries, two[1] recovered without operation and two[2] after tracheotomy. No words can be more eloquent than these figures.

In the Toner Lecture I have reproduced several illustrations of ulceration and necrosis of the cartilages following perichondritis. To these, through the kindness of Dr. M. H. Fussell, of Philadelphia,[3] I am enabled to add another from a photograph of the specimen (Plate III). The case was one of perichondritis of the cricoid, and shows well the necrosed cartilage.

[1] Hérard, quoted by Trousseau, Clin. Med. Syden. Soc. Trans., 2d ed., ii, 407; and Schiffers, Annales Soc. Méd. Chir. de Liège, in Jour. Amer. Med. Assoc., 1884, 70.

[2] Türck and Lüning, Case II, p. 223.

[3] Jour. Amer. Med. Asso., July 3, 1897.

PLATE III.

Dr. M. H. Fussell's case of necrosis of the cricoid cartilage (*a*, *a*) after typhoid fever.

Cartilage Involved.—This was in—

The cricoid,	43 cases.
The arytenoids,	32 "
The other cartilages,	7 "
	82 cases.

In 13 of these both the arytenoids and the cricoid were involved.

In Lüning's table the result is analogous:

Thyroid,	2 cases.
Thyroid and cricoid,	5 "
Thyroid, cricoid, and arytenoids,	3 "
Cricoid alone,	22 "
Cricoid and arytenoids,	14 "
Arytenoids alone,	9 "
	55 cases.

As is pointed out by Lüning, the broad posterior plate of the cricoid is the most frequent portion of this cartilage to be involved, and very frequently the disease extends from the arytenoids, or from their articulation with the cricoid, to the latter.

Laryngoscopic Examination.—In my two tables there were only 14 such examinations made. Lüning has gathered 18, and records the following results:

Ulcers on the vocal chords, processus vocales, posterior laryngeal wall, etc.,	8
Swelling or bulging of anterior surface of posterior wall,	3
Abscess above right vocal chord,	1
Suppurative coating of the vocal chords,	2
Subglottic symmetrical fixed red swelling (chorditis vocalis inferior hypertrophica (?) perichondritis of the cricoid (?)),	2

In all cases there was redness, swelling, edema of portions of the larynx, rarely of the whole larynx, but chiefly of the chords, epiglottis, posterior wall, arytenoids, and their mucous membrane.

Prognosis.—Of 197 cases of all kinds of stenosis in which the result is given, 132 died and 65 recovered—a mortality of 67 per cent. Bad as is this showing, it is far more striking when we separate the cases which were tracheotomized from those which were not. Thus:

Of 98 cases *not* operated on, 21 recovered and 77 died—a mortality of 78.6 per cent.

Of 99 cases operated on, 44 recovered and 55 died—a mortality of only 55.5 per cent.

In necrosis of the cartilages, the most dangerous form of stenosis, as already stated, the mortality was almost 95 per cent.

To add to the eloquence of the contrast between 78.6 per cent. without operation and 55.5 per cent. with operation, I may quote the following from my Toner Lecture:

"When it is remembered that in two of the fatal cases the larynx was not opened, though tracheotomy was apparently performed; and a third, in full recovery thirteen days after the operation, on the removal of the cannula was suddenly suffocated before it could be replaced; and in another the cannula became displaced in front of the trachea [that in over half of Lüning's 14 original cases goiter was a serious complication and several times was the direct cause of death]; that in many, if not in most, of the cases the operation was deferred until the last possible—that is, the most unfavorable—moment;

that many cases that might have been rescued were plainly *allowed* to die from exhaustion, or even from positive suffocation, by timid doctors, cases in which the result could not have been worse had an operation been performed;—the question of operation would seem to be decided."

All that I said twenty years ago as to the need of an *early* tracheotomy the moment that the existence of perichondritis is recognized and suffocative attacks occur is only reinforced by the larger results from the later added cases. Delay can not be entertained for a moment, for it means a speedily fatal result. The terrible facts are before us. Let us hope that the lesson they teach will be learned.

Intubation.—Since my former lecture intubation has been introduced as an operative procedure. I know of no case in which it has been practised, but it may well claim attention in the few cases to which it may be applicable. In perichondritis it would evidently be worse than useless to employ it in place of tracheotomy. It affords no means of escape for the pus, the necrotic tissue, or the fragments of the cartilage. But in the rare cases of simple edema, and in some of ulceration, its use should be considered, though I fear that it would rarely afford the relief which tracheotomy gives.

Final Results.—Lüning has collected the most careful and complete statistics of the final results as to whether the cannula can be dispensed with and as to the value of later attempts to overcome the stenosis by dilatation or other means.

Of 60 cases which recovered after perichondritis, 11 were able to dispense with the use of the cannula in

periods varying from seven months to six years, but the other 49 were obliged to wear the cannula permanently; some could breathe freely without it during the day but were obliged to wear it at night. One patient was known to have worn the cannula for forty years.

Various means have been employed to get rid of the stricture, especially by dilatation with bougies and special cannulas. But the treatment, especially by dilatation, is wearisome, both to the patient and the surgeon, and rarely has done any good. The special cannulas do not seem to have been any more successfully employed. In a half dozen cases attempts have been made to better the patient's condition by a total splitting of the larynx and the adaptation of a special cannula. These seem to have been at least partially successful, but the cannula had to be worn permanently. For the details the reader is referred to Lüning's paper, page 329.

To the above remarks, which interest the surgeon, the physician, and the laryngologist, I may add, as of especial interest to the last, that a number of cases of *paralysis of the muscles of the larynx* have been reported. Those interested in this aspect of the subject will find it well described by Przedborski.[1] Boulay and Mendel[2] have reported 17 cases, and Bernoud[3] reports another case.

[1] Ueber Lähmungen der Kehlkopfmuskeln beim Unterleibs und Flecktyphus. Sammlung klin. Vorträge, Neue Folge, No. 182, 1897.

[2] Des Paralysies Laryngées dans la Fièvre Typhoïde, Rev. de Laryngol. et Rhinol., 1895, xv, 615.

[3] Lyon Méd., March 28, 1897, p. 453.

CHAPTER XIII.

TYPHOID AFFECTIONS OF THE PLEURA, LUNGS, AND HEART.

ONE of the very first regions in which the pyogenic power of the typhoid bacillus was proved was in the pleural cavity. In 1885 Rendu and de Gennes,[1] and in 1887 A. Fraenkel,[2] found pure cultures of the typhoid bacillus in the pus from cases of purulent pleurisy.

Dr. Westcott has gathered together in all nine cases. In five of them bacteriological examinations showed the bacillus of Eberth.

Except one case, in which the date of onset is not given, every one arose after the third week, and in five from one to two months after the fever, thus illustrating again the late period of these typhoid sequels.

In one[3] it arose from a pneumothorax, occurring on the thirty-eighth day after convalescence, both from the fever and from a broncho-pneumonia. In one case there was an abscess of the lungs,[4] in another gangrene of the lung and abscess of the spleen,[5] and in a third[6] there was pus in the anterior mediastinum.

[1] La France Méd., 1885, ii, 1821.
[2] Verhandl. Sechste Kongress inner. Med., 1887, 179.
[3] Rendu, La France Méd., 1885, ii, 1809.
[4] Ramsey, Annals of Surgery, Jan., 1890, 39.
[5] Griesinger, Infectionskrankh.
[6] Barr, Liverpool Med.-Chir. Jour., 1893, xiii, 346.

Only two cases died, one of abscess of the lung and one of uncomplicated empyema.

The *symptoms* do not call for special mention, but the *treatment* does. At least three of the cases recovered after puncture, including even the one with pus in the mediastinum. In three others incision and drainage, in one of which two ribs were resected, accomplished a cure. The fact that the patient was exceedingly weak led to the choice of aspiration in the mediastinal case just mentioned, and 70 ounces of pus were withdrawn. Even should aspiration not be followed by cure, it may give important temporary relief, especially if the amount of fluid (as in that case) is such as to embarrass the respiration or circulation, and may thus give the patient time to recuperate his forces for a later and more radical operation if that proves to be necessary. In one case[1] two aspirations were needed and it was observed that the typhoid bacilli from the second puncture were less virulent than those from the first.

Five were males and four females; four were under twenty years old and four from twenty to thirty-three; all arose after the third week and two as late as two months from the onset of the fever.

Heart.—There are also recorded two cases of purulent pericarditis[2] and one of an abscess in the wall of the heart. Of the former, one was found at the autopsy, the patient, a boy of nine, dying on the seventeenth day. The latter[3] was in a man of

[1] Weintraud, Berlin. klin. Wochen., 1893, xxx, 345.

[2] Zeller, La France Méd., 1881; and Le Clerc, La France Med., 1881, 54.

[3] Drignet, quoted by Michon, Contrib. à l'Étude des Suppur. dans la Fièvre Typhoïde. Thèse de Lyon, 1880.

thirty-six, who died on the thirtieth day, and was likewise found at the autopsy.

In view of the so frequent and characteristic changes in the heart muscle, it is rather singular that rupture of the muscular fibers, hematomata, and abscess are not more frequent in the heart. Save this one case, they are conspicuous by their absence. It is to be noted, also, that the typhoid bacillus has been found in the muscular tissue of the heart itself.[1]

The *treatment* of a purulent pericarditis of typhoid origin would differ in no way from one arising from any other cause, except that the fever, if convalescence be not yet fully established, would seriously diminish the chances of recovery.

[1] Chantemesse and Widal, Gaz. Hebd., March 4, 1887, 146. (See also pp. 23 and 59, *ante*.)

CHAPTER XIV.

TYPHOID AFFECTIONS OF THE ESOPHAGUS AND THE STOMACH.

I. Stricture of the Esophagus.—In the extensive tables we have collected no cases of typhoid surgical affections of the esophagus other than "diphtheric," exudative, and ulcerative manifestations have been met with.

But through the kindness of Prof. Osler, of Johns Hopkins University, and of Dr. Frederick A. Packard, of Philadelphia, I am enabled to publish two cases, which, so far as I know, are the only cases of the kind on record. Both of them are cases of stricture of the esophagus. Presumably, they followed upon typhoid ulcers resulting in cicatricial contraction.

The *symptoms* and *treatment* are the same as is proper in similar stenosis from other causes, and call for no special comment.

Case XXXV (Osler).—"Mrs. Mary M. J., age thirty-six, Judith, Union Co., N. C., was admitted September 26, 1897, complaining of inability to swallow solid food.

"There was nothing in her family history of any moment.

"Personal History.—She was healthy as a girl; married at twenty-one; has had no children. She has suffered at times from dyspepsia during the past six or seven years.

"Her present trouble began in January, 1896. Dr. Nance, of Unionville, N. C., writes as follows: 'My first visit to Mrs. J. was January 27, 1896, and from this time until about the end of March she was ill with typhoid fever. The actual time in bed was seven weeks. The original attack lasted three weeks, during which time she had no delirium. About the end of the third week she had hemorrhage from the bowels, and passed about a pint of blood. Then she had a relapse, which was much more severe than the original attack, in which she had delirium of a wandering nature. At about the third week of the relapse she developed a considerable gastritis, with accompanying vomiting, which continued, on and off, for ten or fifteen days. There was at no time any hemorrhage from the mouth or blood in the vomit. When she first came out of her delirium, about the end of the sixth week, she complained of some difficulty in swallowing; at first slight, but with a steady increase as her general condition improved, until, at the end of the eighth week, she was nearly in the condition she was when I sent her to the hospital.'

"According to the patient's statement, she has not been able to swallow solid food since convalescence. The pain, she says, was severe at first; she located it at a spot below the outer third of the right clavicle, but after the passage of the stomach-tube it is relieved, and is lower down, at the right costal border. At times she can swallow water or milk without pain; at other times the pain is severe. She has no vomiting, except the regurgitation of food which she tries to swallow. She has never brought up any blood. On trying to swallow solid food, it goes, as she expresses it, to the "bottom of the swallow," and then comes back again. She can hold it sometimes as long as five minutes. Ever since her convalescence she has been living on milk and

thin soup. She feels well, and has no pain except when she tries to swallow. In drinking milk, she takes it very slowly and only a little at a time. She says that at one time there was complete stoppage, due to coffee grounds, and she could not swallow anything for two or three days.

"Condition on Admission.—Patient is a thin, dark woman. The lips are of fairly good color, the tongue has a brownish fur, and the breath is foul. In the physical examination there is nothing of special moment.

"On the 28th the stomach-tube was passed easily for 44 cm., and then an obstruction was met, which could not be overcome by any of the ordinary bougies.

"On the 30th I passed a filiform bougie not quite two mm. in diameter. It was quite impossible to pass the smallest olive-tipped probang, measuring between five and six millimeters.

"On the 30th it was found impossible to engage a three mm. bougie in the stricture, which stopped it exactly 34 cm. from the teeth. From this date on, daily attempts were made by Dr. Mitchell, the house physician, to pass the larger bougies, and it was not until the 13th that two filiform bougies were passed together. The next day they were passed again, and immediately afterward the smallest-sized olive-tipped probang (No. 18, French). The obstruction could be distinctly felt, and the probang seemed to pass over a sharp edge. From this time on the stricture was easily dilated, and, on the first of November, the olive-tipped bougies Nos. 21 and 23 were passed, and on the next day, since her typhoid fever, she was able to swallow some bread and meat."

[illegible]

Dr. Ray E. Whelan in the Youngstown (Ohio) Hospital for typhoid fever, twelve weeks before his admission to my ward. On writing to Dr. Whelan, I received a very courteous note from him giving me the following facts: The patient went through a typical attack of typhoid fever, during which he had hemorrhages from the bowels and from the stomach or esophagus. He was given sulpho-carbolate of zinc in five-grain capsules, but these had to be discontinued owing to the difficulty experienced in swallowing them. No other trouble with swallowing was noted while the patient was on liquid diet. Before his discharge from the Youngstown Hospital bougies were twice passed, and two strictures were encountered, one of which was impassable.

"On his admission to the Philadelphia Hospital, the following history was elicited: The family history was entirely negative as regards his present trouble. He had malaria a year ago. Denied absolutely venereal history, traumatism, and the swallowing of hot or corrosive liquids, and showed no sign of hysterical tendency. He uses alcohol and tobacco to excess.

"He was very much emaciated, pale, and weak. The tongue was lightly coated, the tonsils a little enlarged, the pharynx very red. Physical examination showed absolutely no other pathological change except in the esophagus. The swallowing time was from twenty-five to thirty-five seconds. A bougie (1.5 cm. in diameter) was introduced and met with an obstruction about 14 cm. from the teeth. On the next day a smaller bougie (7.5 mm.) passed through this stricture with a little persuasion, but at a point 24 cm. from the teeth met with an impassable obstruction. This lower stricture allows now of the engagement of the tip of an esophageal bougie with spindle-shaped bulb, the greatest diameter of the bulb being five mm. He can swallow milk with some difficulty, at times for several hours being unable to

get this through. The passage of the bougie seems to assist swallowing for a time; partly, no doubt, from the fact that large quantities of mucus are expelled after its withdrawal. The patient is still under treatment (Nov. 25, 1897)."

II. Ulceration Causing Hemorrhage or Perforation of the Stomach.—The only surgical complication I am aware of is *hemorrhage* from the stomach; *perforation* is also a possibility;—both due to typhoid ulceration. Pepper[1] refers to such cases, and says: "Typical typhoid ulcers have very occasionally been found in the stomach. They produce no characteristic symptoms, but have been known to cause hemorrhage or perforation." No case of actual perforation has come to my knowledge. Should such a complication arise, the only hope of the patient, as in perforation of the gall-bladder (Chap. XVI) or of the intestine (Chap. XV), is in immediate abdominal section. It is a desperate remedy, it is true, but the only alternative is absolutely certain death. The treatment of the perforation is the same as in other perforations, by inversion of the margins by Lembert's or other suture, provided the circumstances admit of such interference.

While no case of absolute perforation has been found, Soltau Fenwick,[2] records a case to which Dr. Packard has kindly drawn my attention, in which typhoid ulcers very nearly perforated (p. 286), and a second (p. 290, Case 10), in which actual perforation presumably took place, but peritonitis was prevented by the adhesions to the liver. It is to be

[1] Amer. Text Book of Med., i, 91; ii, 769.

[2] Disorders of Digestion in Infancy and Childhood, 1897, p. 286.

noted that the cause of her death was a sudden and severe hemorrhage from one of the ulcers.

Case XXXIII (Fenwick).—"Figure 3 represents a drawing of a stomach taken from a girl, eight years of age, who succumbed during the third week of enteric fever. Four well-defined ulcers were found in the pyloric region, one of which presented a loosely

Fig. 3.—Drawing of the pyloric end of the stomach in a case of enteric fever: *a*, Acute perforating ulcers with clean bases; *b*, an ulcer with adherent slough (W. Soltau Fenwick).

adherent slough. The edges of the ulcers were sharply defined and somewhat undermined, while their bases were situated in the submucous and muscular coats of the organ. On microscopic examination the lymphoid tissue of the stomach was found to be enormously increased, and the supposition that

the ulcers originated in disease of the solitary glands was confirmed by the appearances of the smallest one. From these facts it would appear that under certain circumstances, disease of the solitary gastric glands may give rise to a form of perforating ulcer of the stomach which closely resembles the idiopathic type of the disease."

By the kind permission of Dr. Fenwick and the publisher, the figure is reproduced : Fig. 31.

Case XXXIV (Fenwick).—"A girl, thirteen years old, was admitted into the hospital with the symptoms of typhoid fever of eight days' duration. Vomiting occurred once or twice, but there was no complaint of epigastric pain. At the end of the fourth week of the disease, when the temperature had begun to decline, the patient was suddenly seized with severe hematemesis, after which she became unconscious and died. At the necropsy the anterior wall of the stomach was found to be adherent to the under surface of the liver. Scattered over the inner surface of the stomach there were numerous sharply defined ulcers, the largest of which was about the size of a florin. The edges were thin and undermined and the base formed by the muscular or peritoneal coat. In the first part of the duodenum there was an ulcer of a similar character, while the whole of the intestine, from the jejunum to the rectum, was riddled with typical typhoid ulcers."

Hemorrhage from such ulceration without perforation is rare.

Hematemesis in Typhoid Fever.—Eichhorst mentions having once seen bleeding from the stomach in typhus, and says that Weiss has seen fatal hemorrhage. (*Cf. Case XXXIV.*) There is no reference in the fifth edition (1897) of Eichhorst's

Handbuch der Speciellen Pathologie and Therapie, in the section on the Stomach, under the anatomical lesions, to hemorrhage or perforation. Under the complications hematemesis is mentioned, and the possibility of typhoid ulcer occurring in the stomach is considered.

Through the kindness of Prof. Osler, I am permitted to publish the following cases of hematemesis in typhoid fever.

It is probable that, in general, expectant rather than operative *treatment* would be the wisest course. That two of the three cases recovered without operation would seem to vindicate this view.

The same may be said of *hemorrhage from the bowels*, which, as it is so closely allied to the topic under discussion, may be considered here, though not belonging to the subject of this chapter. It would be extremely difficult to locate the exact point of hemorrhage in the bowel. Even at the necropsy, when the bowel is laid open it is not always easy to find the source of the hemorrhage. How much more difficult it would be to find it when the bowel is closed. The various, and, it may be, the numerous, swollen Peyer's patches would be perceptible to the touch; but how could the surgeon determine which was the one from which the bleeding came? (See Plate IV, Fig. 1.) Resection of all the implicated bowel would not be considered, it seems to me, for a moment; and the large number of recoveries, even after severe hemorrhages from the bowels, would warrant our concluding that the expectant and symptomatic medical treatment is certainly the best.

Case XXXVI (Osler).—"John M., age forty (hospital No. 1683), was admitted August 21, 1890, with a history of illness of some weeks' duration. The chief symptoms were headache and fever. The blood examination was negative. There was a very definite rose-colored eruption. The temperature was never high, not rising above 103°. On the 27th he vomited, and in one of the attacks he brought up a dark greenish-brown fluid containing red blood-corpuscles in a condition of disintegration and a clot of blood about three by two cm. in diameter. On the 29th, 30th, and 31st the stools were very dark in color, and evidently contained blood, and several times he vomited very dark material. He became very anemic, but made a good recovery."

Case XXXVII (Osler).—"Alberta C., colored, age twenty (hospital No. 10,131), admitted June 14, 1894. This patient was admitted in the third week of the disease. On that afternoon she had had a hemorrhage from the bowels. She was bleeding quite freely on admission. Between 6 and 8 P.M. she had five large stools of almost pure blood with clots. Throughout the following day she was extremely feeble; temperature was normal; patient was delirious. On June 16th there was no further bleeding from the bowels. Toward evening the patient was delirious, and her condition was very bad. At 8.15 P.M. she vomited 100 c.c. of dark bloody fluid, which contained blood coloring-matter and red blood-corpuscles. She sank, and died that evening."

Case XXXVIII (Osler).—"Dr. H., age twenty-two (hospital No. 14,933), admitted January 9, 1896 He had a very severe attack, with persistent fever, which resisted the baths. These, though given from the outset, did not check the onset of quite active delirium.

"On January 25th, about the eighteenth day of the disease, the abdomen was a good deal distended,

there was moderate diarrhea, and less delirium. He seemed to be doing very well. He had had no special gastric symptoms. In the afternoon he quite suddenly sprang up in bed and vomited a quantity of dark blood. The amount was difficult to estimate, as it went all over the bed linen. Part of it was collected, and Dr. Parsons estimated the amount to be about 200 c.c. It contained much débris and red blood-corpuscles. The staining on the sheets was quite red.

"On the 26th the temperature was between 103° and 104°, and in the afternoon at 3.05 he vomited between 200 and 300 c.c. of almost pure, bright red blood. The pulse became more rapid, but these two hemorrhages did not appear to have any injurious influence. His temperature gradually fell, and was normal on the 31st. He made an uninterrupted recovery after a most severe attack."

Just as this manuscript is passing through the press I have received from Prof. William Osler, of Johns Hopkins University, under date of January 11th, the following:

Glossitis.—Another very interesting and rare complication of typhoid occurred last week in a convalescent; namely, acute glossitis. The man had an ordinary attack, no fever for ten days; went out on December 31st. He returned three days later with his mouth open and the tongue enormously swollen and very tender. I thought at first it was going to suppurate, but in the course of three or four days it subsided.

CHAPTER XV.

INTESTINAL PERFORATION IN TYPHOID FEVER.

THE frequent occurrence of perforation of the bowel in typhoid fever has long been recognized, but it was not until Leyden,[1] in 1884, first proposed to treat the resulting peritonitis by operative measures, which he believed to be "a most fruitful field for investigation," that surgeons seriously considered the question of its possible success. In the same year appeared an article by Mikulicz,[2] in which he reported three cases of peritonitis which he had treated surgically; one of them being, it is fairly certain, a case of perforation in typhoid fever. In 1886 Prof. James C. Wilson [3] was the first writer in English to suggest that operative measures should be instituted in typhoid perforation. I had the pleasure of being associated with Prof. Wilson in this case, and we discussed most seriously the question of operation, but from day to day decided against its expediency at that time, and the patient happily recovered without operation.

The first operation in a positively known case of typhoid was reported by Lücke,[4] and in the same year Bontecou [5] operated on another patient.

[1] Deutsch. med. Wochen., 1884, 258.
[2] Samml. klin. Vorträge, No. 262.
[3] Phila. Med. Times, Dec. 11, 1886.
[4] Deutsch. Zeit. f. Chir., No. 25, Heft 1.
[5] Jour. Am. Med. Assoc., 1888, No. 10, p. 106.

Before taking up the question of operation, however, there are a few points in reference to perforation itself which will be of value for us to consider. I shall take the figures and some of the tables from the admirable paper of Fitz,[1] in which, with his usual thoroughness and accuracy, he has collected a large number of statistics from tables of 4680 cases of typhoid.

The frequency of typhoid perforation is estimated variously. Thus, Schulz[2] found that peritonitis from intestinal perforation took place in 1.2 per cent. of 3686 Hamburg cases in 1886–87. This accords with the statement of Liebermeister,[3] who found intestinal perforation in 1.3 per cent. of over 2000 Bâsle cases between 1865 and 1872. Hölscher[4] found perforation in 6 per cent. of 2000 cases. Murchison[5] found its frequency was 11.38 per cent. in 1721 cases. In the 4680 cases tabulated by Fitz, the mortality from perforation was 6.58 per cent., which may be accepted, therefore, as fairly representing its frequency.

In 444 cases (Fitz), it occurred among men in 71 per cent. of the cases, and among women in 29 per cent. Certainly, it is much more frequent among men than among women; for what reason we do not know. In children it is very rare.

[1] Trans. Assoc. of Am. Physicians, 1891, vi, p. 200.

[2] Centralbl. f. all. path. Anat., 1891, ii, 289.

[3] Ziemssen's Handb. spec. Path. u. Therap., 1874, ii, 1, 161.

[4] Münch. med. Wochen., 1891, xxxviii, 64.

[5] Continued Fevers, second edition, 1873, 566.

Age at Which Perforation Occurs.—Fitz's table is as follows:

Age.	Cases.	Per Cent.
One to ten years,	7	3.6
Ten to twenty years,	46	23.8
Twenty to thirty years,	77	39.8
Thirty to forty years,	45	23.3
Forty to fifty years,	14	7.2
Fifty to sixty years,	2	1.0
Sixty to seventy years,	1	0.5
	192	

Date of Occurrence of the Perforation.—Fitz's table is, again, as follows:

Week.	Cases.	Per Cent.
First,	4	. .
Second,	32	16.5
Third,	48	24.8
Fourth,	42	21.7
Fifth,	27	14.0
Sixth,	21	13.4
Seventh,	5	. .
Eighth,	3	. .
Ninth,	2	. .
Tenth,	4	. .
Eleventh,	3	. .
Twelfth,	1	. .
Sixteenth,	1	. .
	193	

The Seat of the Perforation (Fitz).—In 167 cases.

Seat.	Cases.	Per Cent.
Ileum,	136	81.4
Large intestine,	20	12.9
Vermiform appendix,	5	. .
Meckel's diverticulum,	4	. .
Jejunum,	2	. .

Hawkins,[1] in 72 cases of perforation, found 61 in the ileum, 3 in the cecum, 3 in the appendix, and 5 in the colon. Adding 5 cases of perforation of the colon from other sources, of the ten cases there were 2 in the ascending, 1 in the transverse, and 7 in the descending colon—of which 5 were in the sigmoid flexure. If, therefore, no perforation be found in the ileum, cecum, or appendix, the next most likely point would be the sigmoid flexure.

In one curious case reported by Haegler,[2] in a woman, aged thirty-five, who had a hernia as large as a child's head (the result of a celiotomy performed some years before), on the sixth day after her admission a perforation occurred through the wall of the hernia, followed by a fecal fistula, eventually ⅔ of an inch wide and two inches long. Within ten days three other fistulæ formed near the first one. In spite of her excessive emaciation and a bedsore over the sacrum she insisted upon going home, where she finally recovered and the fistulæ spontaneously closed. The explanation given by the author is that a loop of intestine in the hernia became the seat of typhoid ulceration, leading finally to perforation.

Number of Perforations.—Fortunately both for the patient and the surgeon, if interference is decided upon, the perforation is almost uniformly single, but Fitz reports that there were in 167 cases:

2	perforations in	19 cases
5	" in	3 "
4	" in	1 case
Several	" in	4 cases
25 to 30	" in	2 "

[1] Lancet, 1893, ii, 245. [2] N. Y. Med. Jour., July 17, 1897, 93, from Correspondenzbl. f. schweiz. Aerzte, 1896, No. 17.

Lebert[1] and Hoffman[2] report the last two cases.

Character of the Perforation.—If the perforation result from the ulceration of a solitary follicle, it is apt to be small and round (Plate IV, Fig. 2; Plate V, Fig. 2). If it result from the perforation of a patch of Peyer, it may be oval or round, and will be much larger than in the former case (Plate IV, Fig. 1; and Plate V, Fig. 1), and may even involve a half of the circumference of the bowel (Plate IV, Fig. 1). Sometimes shreds of tissue will be left partially closing the opening (Plate V, Fig. 1); at others the opening is total and clean cut. Not uncommonly there is a tolerably wide area around the ulcer in which the intestinal wall has been greatly thinned (Plate IV, Fig. 2; Plate V, Fig. 2). This is of great importance from an operative point of view (*vide infra*).[3]

Duration of Life after Perforation.—Of 134 cases (Fitz):

Died on the first day,	37.3 per cent.
" on the second day,	29.5 "
" in the first week,	83.4 "
" in the second week,	9 cases
" in the third week,	4 "
" in thirty days,	1 case
" in thirty-eight days,	1 "

We should naturally expect that perforation would be more frequent in proportion to the gravity of the

[1] Ueber d. Typhus- u. d. Typh.-Epid. d. Jahr. 1857; Friedrich, Die Paracentese d. Unterleibs b. Darmperf. im Abdominaltyph., 1867.

[2] Untersuch. u. d. path. anat. Veränd. d. Organe beim abdominaltyph., 1869.

[3] Plates IV and V are from specimens in the Museum of the Pennsylvania Hospital, which I was kindly allowed to have photographed.

PLATE IV.

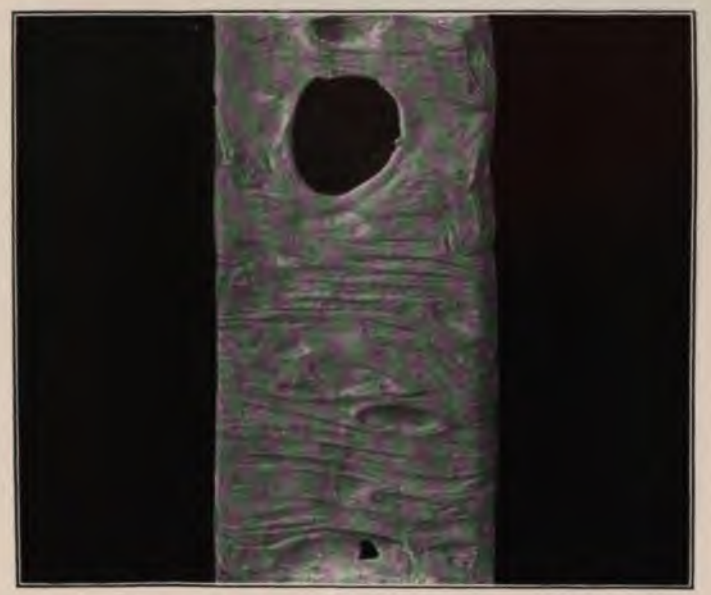


Fig. 1.—Two intestinal perforations in typhoid fever. A third small perforation existed just above the large one. Resection of the bowel would have been the only possible mode of treating the large perforation, as lateral closure would have produced great stenosis. Observe the numerous areas of thinning of the wall of the bowel from ulceration. (Museum of the Pennsylvania Hospital.)

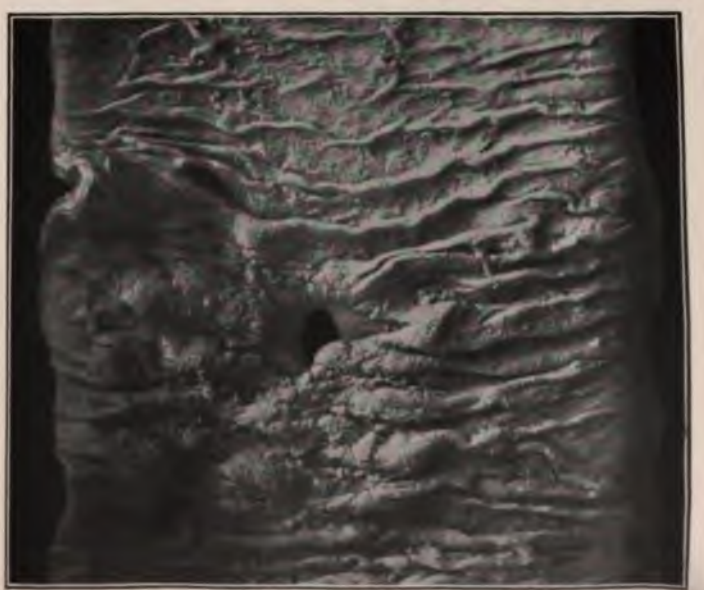


Fig. 2.—Intestinal perforation in typhoid fever. Observe the thinned wall around the perforation. (Museum of the Pennsylvania Hospital.)

PLATE V.

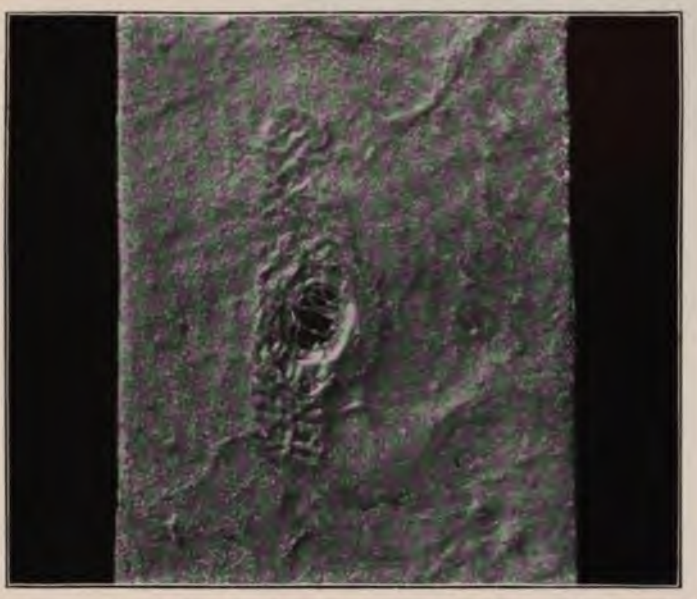


Fig. 1.—Intestinal perforation in typhoid fever. Observe the threads of tissue obstructing the opening. (Museum of the Pennsylvania Hospital.)

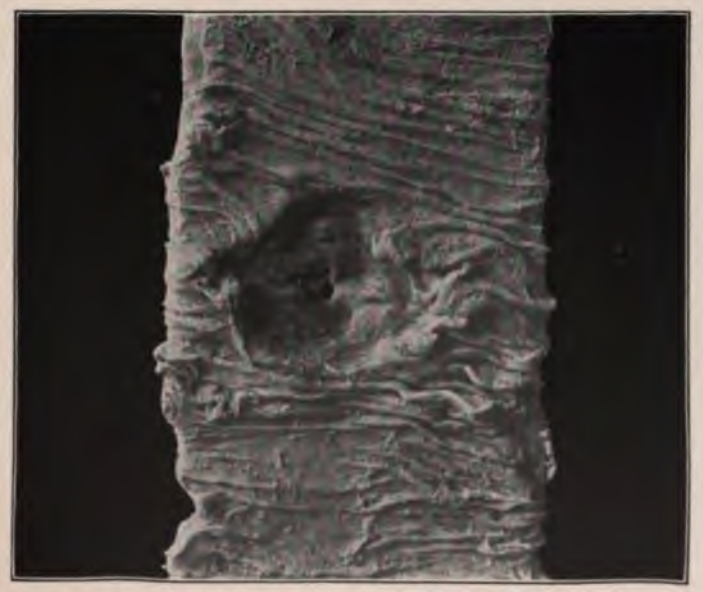


Fig. 2.—Intestinal perforation in typhoid fever. Observe the wide-spread thinning of the intestinal wall around the actual perforation. (Museum of the Pennsylvania Hospital.)

disease, but Fitz states that there is no definite relation between the two. In about one-fourth of nearly 2000 cases the course of the disease was distinctly stated to be mild. Of walking typhoid there were 14 cases, and several cases have been reported in which the perforation was only found at the necropsy.

Diagnosis of Perforation.—Of 80 cases in which a record was made of the symptoms, it was found (Fitz) that in 56 cases the onset was sudden, in 15 the symptoms were gradual or latent, while in five there were no symptoms whatever pointing to the perforation. Armstrong, in a personal letter, says "In none of my cases was the occurrence of perforation indicated by those well-marked, striking symptoms so generally mentioned in text-books."

In addition to this, it must be remembered that fatal peritonitis may arise during typhoid fever without any perforation being discovered, at operation or even by a necropsy. Herringham and Bowlby[1] record a striking case in which the symptoms pointed plainly to perforation, yet the operation revealed only scybala in the colon. The patient recovered.

There are also other causes for typhoid peritonitis than typhoid perforation, as I have shown in the chapters on Abscess (from rupture of the mesenteric glands, and abscesses of the abdominal wall), on the Spleen, and especially in the chapter on the Liver and Gall-bladder, from perforating ulcers of the gall-bladder. Fitz has well said (p. 207), "Since perforation of the intestine in typhoid fever may take place without any suggestive symptoms, and since

[1] Brit. Med. Jour., 1897, i, 265.

suggestive—even so-called characteristic—symptoms may occur without any perforation having taken place, it must be admitted that recovery from such symptoms is not satisfactory evidence of recovery from perforation." But there are certainly a number of cases on record in which perforation could not be doubted which have recovered. Murchison, however, estimates that the mortality of typhoid perforations in general is about 90 per cent., and in those in which general peritonitis supervenes after such perforations it is 95 per cent.

The ordinary *symptoms* which indicate perforation are, especially, sudden and severe abdominal pain, usually in the neighborhood of the right iliac fossa or the hypogastrium, collapse, nausea and vomiting, with occasionally a marked fall of temperature. It would be supposed that in consequence of the perforation the intestinal gases would escape, and this would be followed by disappearance of the hepatic dulness; but this is only rarely reported.

One very important symptom, which has been observed only of late, is the presence or absence of leucocytosis. According to Thayer,[1] there is no increase in the proportion of the white blood-corpuscles during the fever, but rather a slight diminution in their number, which gradually diminishes until convalescence. During the fever, the number may fall even below 2000, and sometimes below 1000, per cubic centimeter. The lowest count seems to be about the end of the third week. Sometimes the white blood-cells increased markedly in number (leucocytosis) with the fever, even without any complication. Four cases

[1] Johns Hopkins Bulletin, iv, p. 83.

were observed by Cabot in which the count was over 11,000, and ran as high even as 17,700 without any other than the typical typhoid lesions. But the effect of complications is very marked and undoubted. I quote the following examples in seven cases, two of typhoid perforation, from Cabot's admirable book [1]:

Perforation. . Case I.—(*a*) Five days before perforation, 8300.
(*b*) At time of perforation, 24,000.
Case II.—At time of perforation, 18,500.

Phlebitis. . . Case I.—(*a*) Two days before onset, 6400.
(*b*) At time of onset, 12,900.
(*c*) One week later, 10,100.
Case II.—(*a*) One week before onset, 4800.
(*b*) At time of onset, 16,200.

Otitis Media. . Case I.—(*a*) At entrance, 5300.
(*b*) Mastoid abscess, 16,400.
Case II.—(*a*) At entrance, 8400.
(*b*) Two weeks later, after opening drum membrane (sero-purulent discharge), 11,200.
Case III.—(*a*) At entrance, 7320.
(*b*) Otitis, 14,000.

In addition to this he states that "a freely discharging otitis soon ceases to cause leucocytosis—*e. g.*, a case of serous otitis media seven days after puncture, but still freely discharging, showed but 5320 white cells a cubic centimeter. An abscess of the buttock raised the count from 8000 to 11,200, and a hemorrhage from 8000 to 11,300." General bronchitis and cystitis had usually no such effect. In two cases simulating otitis, with a normal blood count, the trouble turned out to be functional.

[1] Clinical Examination of the Blood, 1897.

It is to be noted, however, that he adds: "It occasionally happens in very exhausted patients that complications fail to produce any leucocytosis, the patient—as in some fatal cases of pneumonia or purulent peritonitis—being unable to react against the infection. . . . . These cases, however, are exceptional." In any case of doubt, therefore, if time allows, a blood count should be made in order to assist us in the diagnosis.

The *result of intestinal perforation* is, in a few cases, a localized abscess similar to the localized abscess which follows a perforative appendicitis; but, in unfortunate contrast with perforative appendicitis, in the majority of cases no such agglutination of the intestine occurs, and, therefore, a generalized suppurative peritonitis follows speedily upon the perforation. This is very natural, since the intestinal contents are more than usually infective, both from the colon bacillus and from the typhoid bacillus as well as the ordinary pyogenic bacteria. If the perforations are multiple, the peritonitis is, of course, so much the more certain to become general. Ordinarily, the typhoid bacilli have not been found in the peritoneum following perforation. Possibly, as suggested by Lannois, they have been overgrown and [illegible] by the more active and vigorous com[illegible]

The *dangers of surgical interference* are unquestionably very great. They can scarcely be stated better than in the frequently quoted words of Wilson, written over eleven years ago:[1]

"Granted that the chances of a successful issue are heavily against you; that the patient is in the midst or at the end of a long sickness; that his tissues are in the worst state to stand the injuries from the knife; that the lesions of the gut may be very extensive; that the vital forces are at the lowest ebb;—no one has yet hesitated to perform a tracheotomy in the laryngeal complications of enteric fever, which require it to save life for these reasons. The operative treatment of purulent peritonitis has been performed many times successfully by the gynecologist in conditions less promising. In point of fact, the objections that may be urged against laparotomy in intestinal perforation in enteric fever are no more forcible than those which would have been made use of at first against the same operation in gunshot wounds of the abdomen. Unfortunately, this question is not to be settled by experiments upon animals. The investigation must be made upon the human subject, where courage to perform it will come from the knowledge that the only alternative is the patient's death."

Commenting on the statement of Fitz, that "the similarity of the symptoms of perforation of the bowel and those of the appendix is striking. . . . The symptoms are not merely similar; they are actually identical, even to the usual localization of the

[1] Phila. Med. Times, 1886, vol. xvii, 177.

consequent peritonitis in the right iliac fossa," Abbe[1] says: "This lucid statement by Fitz must appeal to every observer of appendicitis cases as true to the letter. Why one class of cases should be left to die while we operate on all appendicitis cases, when perforation can be recognized [except, I am sure he would say, when the patient is past hope] does not appear."

But, after all, the appeal must be to the facts in the case. This is the final arbiter. When once physicians are not only on the alert to observe the symptoms of perforation, but when the knowledge that perforation of the bowel can be remedied by surgical means has permeated the profession, so that the instant that perforation takes place the surgeon will be called upon, and if the case be suitable, will operate, we shall find unquestionably a much larger percentage of cures than have thus far been reported. But even at present we have a reasonably large number from which to draw [illegible]. In the table appended to this chapter [illegible]

[illegible]

Under fifteen years of age there were 5 cases and 2 recoveries, or 40 per cent. of recoveries.

In one of these (Case 40) resection of the intestine was done.

If we include (Chap. XVI, Table II) one case of perforation of the gall-bladder with recovery, the number of cases of perforation from both causes is 6, with 3 recoveries, or 50 per cent.

From fifteen to twenty-five years of age there were 23 cases and 3 recoveries, or 13 per cent. of recoveries.

From twenty-six to thirty-five years of age there were 24 cases and 5 recoveries, or 20.8 per cent. of recoveries.

If we include two cases of perforation of the gall-bladder with recovery (Table II), the number of cases is 26, with 7 recoveries, or 26.9 per cent.

Over thirty-five years of age there were 11 cases, with 5 recoveries, or 45.5 per cent. of recoveries.

If we include one fatal case of perforation of the gall-bladder (Table II), the number of cases is 12, with 5 recoveries, or 41.7 per cent.

This discloses the fact that, so far as the present figures go, operations for perforation of the intestine are more fatal between sixteen and thirty-five than under fifteen or over thirty-five.

In Table I the following cases which have appeared in some former tables have not been included for the following reasons:

1. Escher's,[1] since it seems more likely a case of

[1] Wien. med. Wochen., 1887, No. 19, 607.

appendicitis. It is admitted by Finney into his table, though expressly excluded in the text.

2. Greig Smith's.[1] This is stricken out, not only because the diagnosis was doubtful and no post-mortem examination verified the fact, but because Smith himself, in his fifth edition, omits it in his chapter on typhoid perforation.

3. Taylor's,[2] since the diagnosis is extremely doubtful.

4. Price's second case,[3] since, though there was a large amount of filthy contents with fecal odor, the case was more likely tubercular than typhoid, and the report of the case is unfortunately exceedingly meager.

5. The case of Ill, since a personal letter from Dr. Ill assures me that it was not a case of typhoid perforation.

6. L. S. McMurtry's case[4] is rejected, since he writes me that it was a case of appendicitis.

7. A case attributed to Lejars by Monod and Vanverts, as it was not one of typhoid.

8. The case of Steel,[5] as it seems to have been a case of appendicitis at the outset of the fever, and not as a result of perforation during or after the fever.

Some other cases which have been disputed are admitted, as it seems to me that the evidence is sufficiently clear to do so—namely:

1. The case of Mikulicz, No. 1 in the table. This is also admitted by Finney and by Monod and Vanverts.[6]

[1] Abdominal Surgery, 3d ed., 1890, 751.
[2] Lancet, 1890, i, 961.
[3] Med. and Surg. Reporter, Nov. 7, 1891, 577.
[4] Personal letter to the author.
[5] Brit. Med. Jour., 1895, i, 13.
[6] Rev. de Chir., 1897, 169.

2. The cases of Hahn, Nos. 9 and 10, since the statement is direct and positive, though for any other purpose than the fatal result they are of no value.

3. The case of Netschajew and Trojanow, No. 26. Though the case is somewhat doubtful, yet the burden of proof seems to be in its favor, and it is admitted by both Finney and by Monod and Vanverts.

4. The case of Dandridge, No. 28, though the perforation was not found, yet gas and stinking pus were found free in the belly. This is also admitted by Finney.

5. The case of Ferraresi, No. 31. This, also, is a somewhat doubtful case, but, on the whole, with Finney, I would admit it, though it is rejected by Monod and Vanverts.

6. The cases of Hill and Murphy, Nos. 40 and 41, on the basis of a personal communication from Dr. Murphy.

Let us now, turning to the table, see what the results have been, and what conclusions, both as to the methods and time of operation, will show us as to its advisability.

First, the *time of operation* should be wisely chosen. The best time is not during the immediate primary shock which lasts during the first few hours. Happily, in fact, it is very rarely the case that operation *can* be done within several hours after perforation, since, the case being under the care of a physician, it requires time to obtain a consultation with the surgeon, and, when the diagnosis has been reached, still further time must elapse before suitable preparations for operation can be made. The table on page 227 shows that the *second twelve hours*

after perforation, all things considered, has been the most favorable up to this time. Abbe well says that it is essential that "the surgeon should never be so hasty in getting at his work that he enters upon it handicapped by poor assistants, poor light, poor arrangements for irrigation and sponging, or inadequate plans for restoration from shock." The earliest moment at which the operation can be done after the immediate shock of the perforation, provided, of course, there has been any, as is sometimes not the case, the better it will be for the patient. Every hour then counts, since the infection of the peritoneum becomes more diffuse and more intense.

On the other hand, no prudent surgeon would decide to operate in a case in which all the conditions, both general and local, forbade the hope of a recovery. The only contraindication recognized by Van Hook[1] is that the patient is moribund, but I can well imagine that, as in perforation from other cause, the general state of the patient, the pulse, the temperature, and the degree of collapse and of exhaustion will all count as essential factors in formulating a conclusion. Naturally, the cases operated on after relapse or toward the end of the acute febrile attack will result better than those in which the perforation occurring earlier requires the patient not only to recover from the operation, but to have strength enough to carry him through the fever which has not as yet subsided. Yet Finney has called attention to the remarkable vitality exhibited by some of the cases that recover, and states

[1] N. Y. Med. Jour., Nov. 11, 1899, p. 621.

of three cases "who subsequent to the operation suffered two relapses, one, of great severity, had suppurating otitis media, left-sided pleurisy, had right-sided femoral phlebitis, severe neuritis of both legs, and the painful toes so common after the cold bath treatment," and yet recovered.

An analysis of the cases in which the *interval between the onset of the symptoms and the time of operation* is stated shows us the great danger of delay. This is stated in 60 cases as follows:

	Cases.	Recovered.
Within twelve hours,	15	4
Twelve to twenty-four hours,	20	6
Twenty-four to forty-eight hours,	13	1
Two to three days,	6	2
Three to four days,	4	0
Five days,	1	0
Thirty-eight days,	1	0
	60	13

In the cases operated on within twelve hours, the percentage of recoveries was 26.7 per cent.; between twelve and twenty-four hours, 30 per cent. After twenty-four hours the mortality was total, except one after twenty-six hours (No. 64) and the two that recovered between two and three days (Nos. 25 and 28). The last two cases we must consider as exceptional and not impairing the rule that *if the operation is not done within about twenty-four hours after the perforation there is practically no hope of a recovery.*

Technic of the Operation.—The incision may be either median or lateral. If the case, by percussion, is fairly well proved to be an encysted localized peri-

tonitis, I would very decidedly prefer the lateral incision, just as in an appendicitis with a similar condition the lateral incision is preferable. If, however, the case is one of general peritonitis, which in typhoid perforation is far more frequent than the localized form, the incision would best be made in the middle line, since it offers far better opportunities for cleansing the peritoneum, and gives us ready access to the ileum, which is the usual seat of the perforation. It should be a free incision, long enough for the necessary manipulations.

Our first object is to find the perforation. As already shown (p. 214), this is usually in the ileum; but if not there, then the cecum, the appendix, and the sigmoid flexure of the colon should be examined. In the jejunum perforation is very rare, and in the duodenum it may be said almost never to take place. Sometimes the search will be facilitated by cleansing the abdomen of the quantities of fecal matter and foul pus which coat the intestines; sometimes the perforation may be readily discovered without this. If flushing is done it would be best by the sterile salt solution, though Abbe used with advantage a very weak bichlorid solution, 1 : 20,000. As soon as a perforation is discovered, it should be sutured. Sometimes the edges have been pared, but I see no reason for this, as for effective closure we must depend upon the peritoneal adhesions. To trim the edges is both a waste of time and tissue. But in case two perforations are contiguous, it may well be that excision would best prepare the bowel for suture. All of the thinned intestinal wall surrounding the perforation (see page 216 and Plates

IV and V) should be turned in, if this will not produce too much stenosis of the bowel.

In five of the cases tabulated (Nos. 31, 40, 70, 72, 73), there was such wide destruction of tissue that a portion of the bowel was resected. If this condition is found, no other course is open to the surgeon except, it may be, the establishment of an artificial anus, which may be closed later. As a general rule, I rather prefer the latter course; but it is an encouragement to us to find that the first two of the cases of resection recovered. An artificial anus in the ileum I have found in cases other than typhoid to be objectionable, since the intestinal juices are very irritating to the skin.

A continuous suture is decidedly not the best. The tissues of the bowel are very frail and may readily tear out, and, if the suture be continuous and it tears out at one point, it weakens the whole line of suture. Independent mattress sutures, as advised by Halsted, are the best, not only because, as Abbe found, they do not so readily tear the friable tissue, but also because they shorten the operation, since there is but one knot to be tied for every two sutures. Whether one, two, or three rows of sutures shall be applied depends on the case, but we must remember that every additional row narrows the lumen of the bowel. As soon as one opening has been closed, a search should be made in the regions above indicated for either a second perforation, or possibly even more than two, and also for any suspicious weakened spots which show that other perforations are imminent. In cases 51 and 60 subsequent perforations destroyed life in

cases which were apparently recovering, and in case 62 had life been prolonged a little while a new perforation would have occurred and ultimately killed the patient. In two other cases (40 and 48) such suturing at points of impending perforation probably saved both lives.

How far the search for multiple or impending perforations shall go must be decided by the operator in each case. Very prolonged search over a large portion of the intestine involves both time and added shock. Most of the perforations occur within six feet, and especially within the first two or three feet of the ileo-cecal valve. If none are found within this distance nor in the other regions of probable perforation (*vide supra*), I think, as a rule, it would be safer not to prolong the search further. Fewer lives would be lost by such abbreviation of the operation, and by avoiding the necessary mechanical injury and cooling of the intestine, than by missing a very exceptional second ulcer at another point. Whether we shall only flush the abdomen, or whether we shall clean it by wiping, is, I think, to be decided very much by later experience. Finney[1] has made a strong plea for systematic wiping of the bowel with pledgets of gauze wrung out of a hot salt solution in suppurative peritonitis from whatever cause.

In no case, it seems to me, should the abdomen be entirely closed. Drainage should be the rule. If peritonitis arise from other cause than typhoid perforation, scarcely any surgeon would think of closing the abdomen; and the same rule should hold good here. Moreover, I think well of

[1] Johns Hopkins Hosp. Bull., July, 1897.

packing the wound with iodoform gauze, and I would prefer that the point or points of suture in the bowel should be placed, if possible, immediately below the gauze packing, so that if a fecal fistula formed, as occurred in Abbe's case (No. 25), there should be a ready and unobstructed egress for the intestinal contents. In spite of the bad condition of the patients, a number of post-mortems have shown that the wounds become agglutinated and heal as well as other similar wounds in the intestine. In case after case of those that died the post-mortem showed that the stitches had held perfectly, and that no later extravasation had occurred, though this took place in Lücke's and Kimura's cases (Nos. 2 and 14). The very fact that such wounds of the intestine heal readily is a great encouragement.

The cause of death is commonly the septic condition or the profound exhaustion which has been produced by the fever; to which the operation, unfortunately, as a rule, can but add something.

In every case, before the abdomen is closed the appendix should be sought and examined. Typhoid ulcers are sometimes found in the appendix. This part of the intestinal tract, therefore, should never be overlooked. If true appendicitis is present, as may occasionally happen, or if a typhoid perforation exist there, the appendix should, of course, be removed. This was done in several of the cases in the table. If, however, the appendix be not diseased, it should not be removed, as it adds to the length of an operation in which every minute counts.

I have recently operated upon two cases of ap-

pendicitis in which the first attacks followed shortly after typhoid. As in neither case were the typhoid bacilli found in cultures from the contents of the appendix, the connection between the two diseases seems to be accidental and not causal. In the last case, over three years after the typhoid attack, the blood reacted to the Widal serum-test; the ileum showed several oval white patches, corresponding to Peyer's patches, and the mesenteric glands were greatly enlarged. The bacteriological examination showed the colon bacillus and the micrococcus pyogenes albus in a tube inoculated from the peritoneum, the colon bacillus and micrococcus fœtidus in the tube inoculated from the interior of the appendix, and the micrococcus pyogenes albus in pure culture in the tube inoculated from a mesenteric gland which I excised. The gland itself, Prof. Coplin reports, showed areas of coagulation necrosis with softening. In spite of the infection of the peritoneum, the patient made a perfectly smooth recovery from the operation, but still has a hectic temperature —suggestive of tuberculosis, though there are no tubercle bacilli in the sputum.

Unfortunately, in Case 44 intestinal obstruction followed the operation and was the cause of death. I can scarcely think that we would ever be justified in re-opening the abdomen in such a case. Possibly, a very exceptional case might justify such a procedure, but a typhoid patient rarely escapes with his life, even after one operation, and could not be expected to survive a second. The same remark would apply to any new perforation which might occur. Such cases must, unfortunately, be left to

their fate, but if the surgeon has been careful to search for and suture any impending perforation, he has done much to prevent such a disaster. In Cases 40 and 48 the suturing of such an impending perforation undoubtedly saved both lives.

If there be such distention of the intestines that it is difficult, if not impossible, to replace them, they should be incised, and, after the gas has escaped, sutured. This was done in several cases.

Mr. Gairdner, Assistant Physician of the Belvidere Fever Hospital, in analyzing 47 cases of peritonitis in typhoid fever with reference to surgical interference, in a very careful and judicious paper in the Glasgow Medical Journal, February, 1897, page 67, reaches the following conclusions, which well express my own feelings, and it is all the more worthy of consideration as the opinion of a thoughtful physician rather than that of an over-sanguine surgeon: "The treatment of peritonitis in the course of enteric fever by laparotomy has hitherto had a moderate success. There is every reason to believe that greater success is possible, and in any case the results are better than those of any other treatment. Laparotomy offers a fair chance to about 49 per cent. of cases, while 19 per cent. of the whole would certainly have a good chance. Nothing but experience can determine what the results will be—better or worse than might be expected *prima facie*. If there is a good cause at least for attempting surgical interference, it becomes incumbent on the profession to afford every facility for making the attempt. This, of course, applies particularly to authorities responsible for hospitals in which enteric fever is treated."

TABLE I.—EIGHTY-THREE OPERATIONS FOR INTESTINAL PERFORATION IN TYPHOID FEVER.—*Continued.*

Reporter.	Reference.	Age.	Sex.	Location and Character of Lesion.	Date of Occurrence.	Operation.	Interval before Operation.	Result.	Remarks.
[19] 1892. W. Korte.	Arch. f. klin. Chir., 1892, 44, 646.	17	M.	Lower part of ileum.	About 19th day.	Pus and gas in belly, but no perforation found.	96 hours.	D.	Death 15 days after. Autopsy showed perforation as stated.
[20] 1893. J. E. Thompson.	Trans. Texas State Med. Assoc., 1893, p. 266.	36	M.	Twelve in. above valve, oval ¼ in. × ½ in.	During course.	General peritonitis.	96 hours.	D.	Death 11 hours after. Had not been confined to bed until peritonitis began.
[21] 1893. Bell.	Pers. comm. to J. E. Thompson, Med. Chron., 1895, p. 401.	. . .	. . .	. . .	. . .	Perforation closed. Appendix removed.	48 hours.	D.	Death 62 hours after.
[22] 1893. J. B. Murphy.	Personal communication.	31	M.	Twelve in. above valve. Exudate and adhesions over whole right half of belly.	Eighteenth day.	Lembert-Czerny suture. Flushing. Drain.	41 hours.	D.	Death 3 hours after. Autopsy refused. Collapse 36 hours after accident.
[23] 1894. Cayley and Bland Sutton.	Brit. Med. Jour., 1894, i, 578.	25	M.	Twelve in. above valve.	Twenty-fourth day.	Ulcer excised by oval incision, the mucosa being drawn together by continuous silk suture. Lembert suture through peritoneal coat.	5½ hours.	D.	Death 6 days after. Sutton thought in a subsequent case it would be better to attach perforation to abdominal incision and leave fistula to be dealt with later.
[24] 1894. Allingham.	Brit. Med. Jour., 1894, i, 578.	. . .	. . .	. . .	. . .	Tissue about perforation so rotten that it was brought to surface of belly and stitched in the wound.	. . .	D.	Mentioned in discussion on preceding case.
[25] 1894. Abbe.	N. Y. Med. Rec., 1878, Jan. 5, p. 1.	21	M.	Gangrenous perforation, ¼ inch in diameter, in a Peyer's patch.	About 21st to 28th day (after 3 weeks' illness).	Two pints of foul extravasation. Perforation closed by interrupted and Halstead mattress stitches. Incision tamponed with iodoform gauze.	60 hours.	R.	. . .
[26] 1894. Netschajew and Trojanow.	Bolnitschnaia gaseta Botkina, No. 23, 1894. Med. News, 1894, lxv, (69).	25	M.	Eight in. above valve; pin-point opening.	About 21st to 28th day (after 3 weeks' illness).	Cavity filled with sero-purulent and flocculent exudate. Elliptical piece containing perforation removed. Closed by Czerny-Lembert sutures. Packed with dry sterile gauze.	17 hours.	R.	Diagnosis by exclusion. Condition of Peyer's patches not mentioned.

[27] 1894. Alexandroff.	Report Hosp. of St. Olga, in Moscow, 1890, p. 198.	9	M.	Three large perforations in appendix.	On 35th day.	Peritonitis. Appendix removed. Lips of stump sutured with silk. Incision closed with silk, and gauze drain.	36 hours.	D.	Death ½ an hour after. Diagnosis confirmed at autopsy.
[28] 1894. Dandridge.	Personal communication.	9	M.	Perforation not found.	Twenty-first day.	Gas and stinking pus free in cavity. Appendix not found in hasty search. Operation to relieve distention of belly. Washed out and drained.	After 48 hours (on 3d day).	R.	. . .
[29] 1894. Hare.	Intercolonial Quarterly Journal, 1895, Feb.	. . . .		Six and 8 in. above valve.	. . .	Abscess between coils of intestines and pelvis. Ileum incised to give exit to gas. General peritonitis. Lembert sutures. Flushing. Glass drain.	. . .	D.	Death 18 hours after. Perforations impervious to water under pressure.
[30] 1894. B. F. Kingsley.	Pers. comm. to J. M. T. Finney, Ann. of Surg., March, 1897, p. 233.	16	F.	In ileum; "diameter of a lead pencil."	About 12th day (in 2d week).	Cavity contained considerable cloudy serum and small clots. Edges of perforation pared and sutured with Lembert sutures. Irrigation.	15 hours.	D.	Death 5 hours after. No autopsy.
[31] 1894. Ferraresi.	Bull. de Soc. Lancisiana d. Osp. di Roma (1895), 1896, xv, 1, 9-14.	24	F.	In ileum, opposite mesenteric attachment.	Sick only 4 days.	Foul feculent pus in cavity, and ulcer in ileum. Intestine resected. Irrigation; gauze drainage.	. . .	R.	Examination of portion resected showed absence of tubercle bacilli and epithelial or connective-tissue elements. Diagnosis of typhoid made on these grounds.
[32] 1894. Canoli.	Cited by Ferraresi, *loc. cit.*	. . . .		. . .	. . .	. . .	. . .	D.	Said to have operated on two cases *in extremis*, and both died. Autopsy in both cases confirmed diagnosis.
[33] 1894. Canoli.	Cited by Ferraresi, *loc. cit.*	. . . .		. . .	. . .	. . .	. . .	D.	
[34] 1894. Damer Harrisson.	Brit. Med. Jour., 1894, Oct. 20.	. . . . .		Two perforations.	(About 18th day.) In 3d week.	Symptoms not distinct until 36 hours after first indication. Suture. Irrigation boric acid solution.	36 hours +.	D.	Death 17 hours after.
[35] 1894. Termet.	In Barbe, Thèse de Paris, Ob. 20. Cited by Monod and Vanverts. Rev. de Chir., 1897, xvii, 169.	21	F.	. . .	(Three weeks after beginning.) About 21st day.	Suture of a perforation. Lavage with sterilized water. Drainage.	48 hours.	D.	Death several hours later.
[36] 1895. Ricketts.	Cincin. Lancet-Clinic, 1895, April 6, p. 353.	35	M.	No perforation seen, but fully two quarts of pus found in belly. Intestines matted, appendix healthy.	In third week.	. . .	72 hours.	D.	Death 11 hours after. Onset sudden, after straining at stool. Case seen in consultation, and hence the delay in operating.

TABLE I.—EIGHTY-THREE OPERATIONS FOR INTESTINAL PERFORATION IN TYPHOID FEVER.—*Continued.*

Reporter.	Reference.	Age.	Sex.	Location and Character of Lesion.	Date of Occurrence.	Operation.	Interval before Operation.	Result.	Remarks.
[37] 1895. Parkin.	Brit. Med. Jour., 1895, i, p. 192.	32	F.	Twenty-four in. above valve.	About 21st day.	Intestine folded longitudinally on itself and sewn with Lembert sutures, continuous. Keith tube. Incision sutured.	2 hours.	D.	Death 3 days after. Became delirious, got out of bed; then abdominal pain, followed by death in 10 hours.
[38] 1895. Laidley.	Amer. Jour. Obs., 1895, Nov., p. 791.	. . .	. . .	. . .	. . .	. . .	. . .	D.	Death 12 hours after.
[39] 1895. J. E. Thompson.	Med. Chron., 1895, Sept., p. 401.	. .	M.	In cecum, which was out of place in left hypochondrium, near spleen. Perforation ¾ in. long.	Ailing a week or two.	Excised affected portion, and fistula established in median incision.	. . .	D.	Death 8 hours after. Autopsy showed 6 other typhoid ulcers in ileum, none deeper than the mucous membrane.
[40] 1895. W. Hill.	Pers. comm. to J. B. Murphy.	13	M.	. . .	Sixth week.	Perforation involved so large a portion of circumference of bowel that resection was made and Murphy button used. An impending perforation sutured and brought near abdominal wound. A fistula occurred here, but closed in 10 days.	12 hours.	R.	Button voided in 17 days.
[41] 1895. J. B. Murphy.	Personal communication.	31	M.	Peritonitis most marked close to the valve.	Eighteenth day.	No perforation found, but much excoriation and injection over base of one ulcer. Irrigation with normal salt solution, and two quarts left in peritoneum, which was sealed.	. . .	R.	. . .
[42] 1895. L. W. Hotchkiss.	N. Y. Med. Jour., Jan. 11, 1896.	24	M.	Five in. above valve.	Early in 3d week.	Peritoneal surface about perforation turned in with Lembert sutures. Flushed with normal salt solution. Gauze packing and drain.	. . .	D.	Death 4½ hours after.
[43] 1895. C. K. Briddon.	Ann. of Surg., 1896, Feb., p. 198.	18	M.	Nine in. above valve.	Fifteen days after being discharged, convalescent, from hospital.	Perforation closed with double row of Lembert sutures. Flushing with normal salt solution.	72 hours.	D.	Death on same day

[44] 1895.	Bogart.	Ann. of Surg., 1896, i, p. 596.	30	M.	Four in. above valve.	Four months after attack. A second attack of fever, of 3 weeks' duration, 3 mos. after first attack, and reappearance of fever 10 days before accident.	Appendix normal, but adherent to a coil of ileum 4 in. above valve. On separating, a perforation found. Sero-pus about this area when abdomen was opened. Running Lembert sutures of fine silk. Appendix removed. Wound partly packed with iodoform gauze.	20 hours.	D.	Death 3 days after. Autopsy showed obstruction of hepatic flexure of colon by old adhesive band.
[45] 1895. Price.	Joseph	Med. and Surg. Rep., 1896, Nov. 7, p. 577.	30	F.	Two perforations, one a few inches above valve, large, irregular, and very necrotic; the 2d 6 in. above and ½ in. in length.	After 3 weeks' illness.	A puddle of filthy fluid found about the perforations, and omentum and appendix involved in adhesions.	. . .	R.	. . .
[46] 1895. Price.	Joseph	Med. and Surg. Rep., 1896, Nov. 7, p. 577.	28	F.	"Multiple bowel fistulæ."	After 3 weeks' illness.	General angry peritonitis, filthy bowel contents and inflammatory products. Thorough irrigation and drainage. Stitched multiple bowel fistulæ.	. .	R.	. . .
[47] 1895.	Watson.	Bost. Med. and Surg. Jour., cxxxiv, 1896, No. 13.	28	M	Loop of intestine adherent to abdominal wall by recent delicate adhesion. In center of adherent area a perforation, size of thumb-end.	Seventh week.	Feces oozing out. Localized peritonitis. Edges of perforation excised and sutured with continuous suture.	12 hours +.	R.	. . .
[48] 1895.	Sifton.	Chicago Clin. Rev., vol. iv, No. 7, April, 1895.	39	M.	Twenty-four in. above valve, $\frac{1}{16}$ in. diameter.	In 4th week.	One pint of liquid feces flowed out of central incision. Two rows Lembert sutures. A second point, 10 in. higher, about to perforate, also sutured. Irrigation. Iodoform gauze drain into pelvis and wound closed about it.	11 hours +.	R.	. . .
[49] 1895.	Lejars.	La Presse Médicale, 1896, Jan. 1.	23	M.	Six in. above valve, circular, size of a 50-franc piece.	Eighteenth day.	Belly contained great quantity of yellowish liquid mixed with fecal matter. Freshened edges and united by 5 interrupted sutures of fine silk. Cleansed with tampons. Mikulicz drainage. Intravenous injections of serum.	24 hours (?).	D.	Death 3 days after. At autopsy perforation found closed. Peritonitis had progressed. [A second case attributed by Monod and Vanverts (Revue de Chirurgie, March, 1897) to Lejars, under the same reference, is not a typhoid case.]

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Reporter.	Reference.	Age.	Sex.	Location and Character of Perforation.	Duration of Disease.	Operation.	Interval between Perforation and Operation.	Result.	Remarks.
[50] 1896. Hoffis.	Lancet, 1896, i, 1284.		M.	Eight in. above valve.	Seventeenth day (?) (first signs of disease).	Gas, blood and feces in peritoneal cavity. Ragged edges excised and sutured by Lembert sutures. Cavity flushed.	Immediate.	D.	Death 36 hours after. Autopsy. Gut near sutured ulcer deeply congested. At lower end of sutures necrosis had extended to peritoneum, but not perforated.
[51] 1896. Routier.	Reported by Dieulafoy, Bull. de l'Acad. de Méd., 1896, 3 S., xxxvi p. 475.	. .	M.	Two in. above valve.	Fourteenth day.	Fecal liquid in peritoneum. Suture by two planes. Incision not completely closed. Iodoform gauze drainage.	24 hours.	D.	Death 10 days after. Two new perforations near the first had occurred.
[52] 1896. G. E. Armstrong.	Montreal Med. Jour., Feb., 1897, p. 661.	18	M.	Six in. above valve.	Tenth to 13th day.	Abdominal cavity contained gas, feces, serum, and seropurulent liquid. Closed by double row Lembert sutures, cavity washed with normal salt solution. Iodoform gauze drainage and glass tube.	18 hours.	D.	Death 40 days after. On 24th day a second perforation, visible through incision. Almost complete absence of reparative power noticeable; wound gaping after stitches removed. On 28th day a third perforation, with considerable loss of blood. At autopsy closure of first perforation was complete.
[53] 1896. G. E. Armstrong.	Brit. Med. Jour., 1896, ii, p. 1621, and personal letter.	27	M.	Ten in. above valve.	End of third week.	Closure found air-tight at post-mortem.	26 hours.	D.	Died in 12 hours.
[54] 1896. G. E. Armstrong.	Brit. Med. Jour., 1896, ii, p. 1621, and personal letter.	18	M.	Eight in. above valve.	Sixteenth day.	. . .	12 hours.	D.	Died in 11 hours.
[55] 1896. G. E. Armstrong.	Brit. Med. Jour., 1896, ii, p. 1621, and personal letter.	28	M.	Six in. above valve.	Thirteenth day.	. . .	18 hours.	D.	Died on 45th day from pelvic peritonitis from two other perforations on the 28th and 35th days.
[56] 1896. G. E. Armstrong.	Brit. Med. Jour., 1896, ii, p. 1621, and personal letter.	. . .	. . .	. . .	. . .	. . .	. . .	D.	[This case and the two immediately following were operated upon by Armstrong's colleagues.]

[44] 1895.	Bogart.	Ann. of Surg., 1896, i, p. 596.	30	M.	Four in. above valve.	Four months after attack. A second attack of fever, of 3 weeks' duration, 3 mos. after first attack, and reappearance of fever 10 days before accident.	Appendix normal, but adherent to a coil of ileum 4 in. above valve. On separating, a perforation found. Sero-pus about this area when abdomen was opened. Running Lembert sutures of fine silk. Appendix removed. Wound partly packed with iodoform gauze.	20 hours.	D.	Death 3 days after. Autopsy showed obstruction of hepatic flexure of colon by old adhesive band.
[45] 1895.	Joseph Price.	Med. and Surg. Rep., 1896, Nov. 7, p. 577.	30	F.	Two perforations, one a few inches above valve, large, irregular, and very necrotic; the 2d 6 in. above and ½ in. in length.	After 3 weeks' illness.	A puddle of filthy fluid found about the perforations, and omentum and appendix involved in adhesions.	. . .	R.	. . .
[46] 1895.	Joseph Price.	Med. and Surg. Rep., 1896, Nov. 7, p. 577.	28	F.	"Multiple bowel fistulæ."	After 3 weeks' illness.	General angry peritonitis, filthy bowel contents and inflammatory products. Thorough irrigation and drainage. Stitched multiple bowel fistulæ.	. .	R.	. . .
[47] 1895.	Watson.	Bost. Med. and Surg. Jour., cxxxiv, 1896, No. 13.	28	M	Loop of intestine adherent to abdominal wall by recent delicate adhesion. In center of adherent area a perforation, size of thumb-end.	Seventh week.	Feces oozing out. Localized peritonitis. Edges of perforation excised and sutured with continuous suture.	12 hours +.	R.	. . .
[48] 1895.	Sifton.	Chicago Clin. Rev., vol. iv, No. 7, April, 1895.	39	M.	Twenty-four in. above valve, $\frac{1}{16}$ in. diameter.	In 4th week.	One pint of liquid feces flowed out of central incision. Two rows Lembert sutures. A second point, 10 in. higher, about to perforate, also sutured. Irrigation. Iodoform gauze drain into pelvis and wound closed about it.	11 hours +.	R.	. . .
[49] 1895.	Lejars.	La Presse Médicale, 1896, Jan. 1.	23	M.	Six in. above valve, circular, size of a 50-franc piece.	Eighteenth day.	Belly contained great quantity of yellowish liquid mixed with fecal matter. Freshened edges and united by 5 interrupted sutures of fine silk. Cleansed with tampons. Mikulicz drainage. Intravenous injections of serum.	24 hours (?).	D.	Death 3 days after. At autopsy perforation found closed. Peritonitis had progressed. [A second case attributed by Monod and Vanverts (Revue de Chirurgie, March, 1897) to Lejars, under the same reference, is not a typhoid case.]

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Reporter.	Reference.	Age.	Sex.	Location and Character of Lesion.	Date of Occurrence.	Operation.	Interval before Operation.	Result.	Remarks.
[50] 1896. Hollis.	Lancet, 1896, i, 1284.	33	M.	Eight in. above valve.	Seventeenth day after first signs of disease.	Gas, blood, and feces in peritoneal cavity. Ragged edges inverted and closed by Lembert sutures. Cavity flushed.	Immediately.	D.	Death 36 hours after. Autopsy: Gut near sutured ulcer deeply congested. At lower end of sutures necrosis had extended to peritoneum, but not perforated.
[51] 1896. Routier.	Reported by Dieulafoy, Bull. de l'Acad. de Méd., 1896, 3 S., xxxvi, p. 475.	. .	M.	Two in. above valve.	Fifteenth day.	Fecal liquid in peritoneum. Suture by two planes. Incision not completely closed. Iodoform gauze drainage.	24 hours.	D.	Death 10 days after. Two new perforations near the first had occurred.
[52] 1896. G. E. Armstrong.	Montreal Med. Jour., Feb., 1897, p. 601.	28	M.	Six in. above valve	Tenth to 13th day.	Abdominal cavity contained gas, feces, serum, and sero-purulent liquid. Closed by double row Lembert sutures, cavity washed with normal salt solution. Iodoform gauze drainage and glass tube.	18 hours.	D.	Death 40 days after. On 24th day a second perforation, visible through incision. Almost complete absence of reparative power noticeable; wound gaping after stitches removed. On 28th day a third perforation, with considerable loss of blood. At autopsy closure of first perforation was complete.
[53] 1896. G. E. Armstrong.	Brit. Med. Jour., 1896, ii, p. 1621, and personal letter.	27	M.	Ten in. above valve.	End of third week.	Closure found air-tight at post-mortem.	26 hours.	D.	Died in 12 hours.
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[56] 1896. G. E. Armstrong.	Brit. Med. Jour., 1896, ii, p. 1621, and personal letter.	. . . .		. . .	. . .	. . .	. . .	D.	[This case and the two immediately following were operated upon by Armstrong's colleagues.]

[57] 1896. G. E. Armstrong.	Brit. Med. Jour., 1896, ii, p. 1621, and personal letter.	. . . .	. . .	. . .	. . .	. . .	. . .	D.	. . .
[58] 1896. G. E. Armstrong.	Brit. Med. Jour., 1896, ii, p. 1621, and personal letter.	. . . .	. . .	. . .	. . .	. . .	. . .	D.	. . .
[59] 1896. Cholzow.	Ann. der Russ. Chir., 1896, Hft. 2. In Centralbl. f. Chir., 1896, No. 42.	24	M.	Eight in. above valve.	Seventh week.	Excision of egg-shaped piece of intestine. Suture. Tamponade. Abdominal wound left open.	4 hours.	D.	Death on 3d day. Was seized with pain after a bath. Had also double pneumonia at time of death.
[60] 1896. Brun.	Société de Chir., Nov. 25, 1896.	14	M.	. . .	Ninth week, on the 14th day of a relapse.	Lembert sutures. Lavage with boiled water. Drainage.	22 hours.	D.	Death on 7th day after. Serum injections. Autopsy showed generalized peritonitis from 5 other later perforations. A plaque of sphacelus on duodenum.
[61] 1896. Brunton and Bowlby.	Lancet, Jan. 30, 1897, p. 312.	37	M.	Center of Peyer's patch; size of a pea.	Eleventh week after onset, and 47th day after a relapse.	Great omentum adherent in pelvis, and intestines beneath adherent and matted together. Half pint of dirty yellow fluid. Old and recent lymph. Ten silk Lembert sutures, transverse to long axis of bowel. Irrigation. Drainage.	18 hours.	R.	Case "unusually well adapted to operative treatment, and could not be compared with cases where the perforation occurred during height of fever." Probably perforation had existed some time and been closed temporarily by adhesions.
[62] 1896. Monod.	Bull. de l'Acad. de Méd., 1896, xxxvi, 550.	35	M.	Twelve in. above valve; small perforation.	Twentieth day.	Sero-purulent liquid in belly, not fetid, lymph on intestines—general peritonitis. Suture in two stages. Also suture over an ulcer threatening rupture. Lavage with boiled water. Iodoform gauze drain.	11 hours.	D.	Death 41 hours after. No fecal matter in peritoneum. An ulcer ready to rupture.
[63] 1897. Potter and Shattuck.	Boston Med. and Surg. Jour., 1897, April 15, p. 354.	19	M.	Twelve in. above valve.	Eighteenth day.	Turbid serum escaped from abdominal incision, of slightly fecal odor. Small perforation closed by double plane of sutures.	. . .	D.	Death 61 hours after operation. Firm union.
[64] 1897. A. C. Panton.	Ann. of Surg., Aug., 1897, p. 219.	45	M.	Twelve to 18 in. above valve. Round ulcer, 5 mm. in diameter, partly closed by lymph.	About 47th day, after convalescence supposed to be well advanced.	Gas and thin yellow fecal matter. Intense general peritonitis. Edges of perforation trimmed, and closed with 15 interrupted Lembert sutures, reinforced by continuous mattress suture. Flushed with normal salt solution. Strips of iodoform gauze. Incision only partly closed. Plastic operation on 38th day to strengthen abdominal wall at wound.	26 hours.	R.	. . .

TABLE I.—EIGHTY-THREE OPERATIONS FOR INTESTINAL PERFORATION IN TYPHOID FEVER.—*Continued.*

Reporter.	Reference.	Age.	Sex.	Location and Character of Lesion.	Date of Occurrence.	Operation.	Interval before Operation.	Result.	Remarks.
[65] 1897. N. P. Dandridge.	Cincinnati Lancet-Clinic, Aug. 21, 1897, p. 177.	19	M.	Twelve inches above valve, $^1_4$ in. diameter.	About 11th day.	Gut covered with lymph for 2 or 3 in. Gas and greenish-brown fluid in belly. Closed with Lembert sutures. Washed with saline solution. Intestine punctured to relieve distention. Loop drawn to incision and packed with iodoform gauze. Strychnin and saline injection.	6 hours.	D.	Death 54 hours after. Purulent peritonitis localized.
[66] 1894. J. M. T. Finney.	Ann. of Surg., 1897, xxv, p. 233.	55	M.	Six inches above valve, $\frac{3}{4}$ in. diameter; sloughing edges.	Twelfth day.	Edges turned in and sutured with Halstead mattress sutures. Cavity cleaned by sponging. Coil irrigated with salt solution. Iodoform gauze drainage.	96 hours.	D.	Death 7 hours after.
[67] 1895. J. M. T. Finney.	*Loc. cit.*	26	M.	Six and a half inches above valve.	Nineteenth day.	Cavity filled with sero-purulent fluid. Peritoneum congested. Flakes of lymph. Halstead's mattress sutures. Intestines punctured for flatus. Gauze drain.	20 hours.	D.	Death 26 hours after.
[68] 1896. J. M. T. Finney.	*Loc. cit.*	47	M.	Fourteen inches above valve, $\frac{1}{8}$ in. diameter; in middle of Peyer's patch.	After week of illness.	Peritonitis, lymph, turbid yellowish fluid. Edges turned in, mattress sutures. Salt solution cleansing. Bismuth gauze strips for drainage.	15 hours.	R.	After otitis media, pleuritis sicca, relapse, thrombosis femoral vein, and furunculosis.
[69] 1895. Geselevitch and Dombrowski.	Laitop. russk. Chir., 1897, ii, p. 407.	36	M.	Two perforations.	Beginning 4th week.	Nine inches of bowel removed. Tamponade. Abdominal wound partly closed.	12 hours.	D.	Death 2 days after.
[70] 1895. Geselevitch and Vanech.	Laitop. russk. Chir., 1897, ii, p. 407.	24	M.	. . .	In 3d week.	Twelve inches of bowel resected. Cavity dried. Tamponade. Wound partly closed.	17 hours.	D.	Death on 7th day. Catarrhal pneumonia.
[71] 1895. Geselevitch and Kadian.	Laitop. russk. Chir., 1897, ii, p. 407.	19	M.	. . .	In 5th week.	Tamponade. Wound partly closed.	120 hours.	D.	Death 3 days later.

[72] 1896. Geselevitch and Vanech.	Laitop. russk. Chir., 1897, ii, p. 407.	27	M.	One, and a second nearly complete.	Fourteenth day.	Twelve inches of bowel resected. Cavity dried. Tamponade. Abdominal wound not sutured.	24 hours.	D.	Death 8 days after, from exhaustion.
[73] 1896. Geselevitch and Vanech.	Laitop. russk. Chir., 1897, ii, p. 407.	28	M.	. . .	Thirteenth day.	Excision and stitches. Tamponade. Abdominal wound not closed.	13 hours.	D.	Death 3 days after.
[74] 1895. Trojanoff.	Laitop. russk. Chir., 1897, ii, p. 277.	29	M.	Four in. above valve.	After 2 weeks. Ambulant.	Peritonitis. Large sero-purulent collection, fecal odor. Egg-shaped excision. Lembert-Czerny sutures. Gauze packing.	16 hours.	D.	Death 14 hours after.
[75] 1891. Zeidler.	Cited by Netschajeff, Bolnitsch. Gas. Botkina, 1894, p. 569.	31	M.	. . .	After 14th day.	Peritonitis. Ichor and fecal matter in cavity. Perforation excised.	3 to 4 days.	D.	Death on same day.
[76] 1893. Lejars.	Sem. Méd., 1896, No. 61, p. 487.	11	M.	Lower part of ileum.	. . .	. . .	. . .	D.	Died next day.
[77] J. A. Hutchinson, of Montreal.	Personal letter.	19	M.	Close to valve.	Thirtieth day.	Edges healthy. Two rows silk Lembert sutures. Saline irrigation. Glass drain.	13 hours.	D	Death in 10 hours.
[78] J. A. Hutchinson, of Montreal.	Personal letter.	19	M.	Six in. from valve.	Seventeenth day.	Much free pus and feces. Two rows Lembert sutures. Saline irrigation. Drain.	4 hours.	D.	Death 12 hours later.
[79] G. E. Armstrong.	Personal letter.	22	M.	. . .	Seventeenth day of a relapse.	Suture. Two other suspicious spots sutured.	29 hours.	D.	Died 38 hours after. Necropsy showed general peritonitis. Sutures tight.
[80] R. C. Kirkpatrick, of Montreal.	Personal letter.	35	M.	Near valve, 2 cm.	Uncertain.	Incision over appendix. Pint of yellow fluid with fecal odor. Intestines covered with lymph. Two rows Lembert sutures, silk. Hot saline flushing, 4 glass drains, and iodoform gauze.	Probably over 24 hours.	D.	Died 3 days after. Culture from abdomen showed intestinal bacteria and a few streptococci. An Italian with no prior history.
[81] Weir.	Annals of Surg., Dec., 1897, xxvi, 707.	40	M.	Eight in. from valve. Size of lead pencil. Second thickened patch near by.	Third week.	Sutures tried, but abandoned on account of thickened wall, tearing out of sutures, and narrowing of lumen. "Catheter introduced into bowel for drainage, and ligature passed through the intestinal wall, securing it *in situ*, and wound packed around this tube."	12 hours.	D.	Died 12 hours after. Temp. 106°.
[82] V. W. Harrison.	North Carolina Med. Jour., Dec. 5, 1897, xli, 368; and personal letter.	18	M.	Eight in. from valve. Size of goose-quill.	Beginning of 2d week.	Edges trimmed. Three rows sutures. Saline irrigation and saline transfusion. Gauze drain.	2 days.	D.	Died in half an hour, from general peritonitis.
[83] H. M. Taylor.	Virginia Medical Semi-monthly, Dec. 10, 1897.	. .	M.	. . .	In convalescence from mild attack.	. . .	About 48 hours.	D.	. . .

CHAPTER XVI.

TYPHOID AFFECTIONS OF THE LIVER AND THE GALL-BLADDER.

I. Typhoid Affections of the Liver.—The intimate anatomical and pathological connection between the intestines, the liver, and the gall-bladder, especially through the portal circulation and the biliary ducts, is self-evident. The commonest cause of abscess of the liver is through an infection following dysentery and similar intestinal diseases. It is not surprising, therefore, that a disorder like typhoid fever, of which the essential lesion is inflammation and ulceration of numerous areas of the intestinal mucous membrane, should occasionally be followed by abscess of the liver. Hölscher[1] found parenchymatous degeneration of the liver in 203 out of 2000 fatal cases—over ten per cent. In view of this fact, and of the thrombosis of the vessels of the liver by the typhoid bacilli actually observed by Fraenkel and Simmonds (Plate I, p. 62), it is rather surprising that so few cases of abscess should be reported, seeing the inevitable exposure of every such patient to the possibility of infection by the typhoid bacillus, the colon bacillus, and the ordinary pyogenic bacteria. Yet in my first series of cases, not one case of hepatic abscess was tabulated. In the second series only 21 cases in all could be

[1] Münch. med. Wochen., 1891, Nos. 4 and 5.

found by Dr. Westcott, the first case being that of Louis, in 1841. A number of cases alluded to by various authors, but not in such detail as to allow of tabulation, are necessarily excluded. Even of the cases tabulated, in one [1] the diagnosis of typhoid is doubtful.

Typhoidal hepatic abscess is in most cases primary. Whether it is the direct result of infection by the typhoid bacillus or not we have too few bacteriological examinations to decide. Dupré [2] obtained a pure culture of the typhoid bacillus from a case of angiocholitis six months after the fever. In another, Rosenberg [3] found the staphylococcus. Most probably, in the majority of cases the pyogenic bacteria and the colon bacillus are the immediate cause, while in a few the typhoid bacillus alone may produce the abscess.

Instead of being a primary infection of the liver, the abscess may sometimes be secondary to typhoid lesions elsewhere, especially if these become infected by pyogenic bacteria. This is really a form of pyemia. Thus, Louis [4] reports the first recorded case, I believe, of hepatic abscess from typhoid, in which it arose after an abscess of the parotid. Romberg [5] refers to two others arising, one from an abscess of the fourth finger, and the second from a thenar abscess. Another case is reported by Chvostek,[6] in which it was secondary to a laryngeal perichondritis.

[1] Daly, Medical and Surgical Reporter, 1882, 346.

[2] Les infec. biliaires, Thèse de Paris, 1891.

[3] Berlin. klin. Wochen., 1890. 192.

[4] Recherches Anat. Path. et Thérap. sur. la Fièvre Typhoide, 1841, 12th ed., p. 118.

[5] Berlin. klin. Wochen., 1890, 192.

[6] Allgem. Wien. med. Zeit, 1866, No 37.

Burder [1] has reported a similar case. In these cases the abscesses are multiple.

Solitary abscesses are more common, but even these are very rare. Langenbuch [2] refers to several cases to which I have not had access, and states that there are on record about 20 cases of typhoidal abscess of the liver. Besides the five cases of multiple abscesses, Dr. Westcott has only found 16 others. To these should be added 12 cases found in the Munich post-mortems by Hölscher,[3] but which are only tabulated so briefly that they can not be used in my statistics.

Of these 21 cases, all died but two. Not a few were only found at the post-mortem, since in the dulled condition of the patient no complaint was made of any hepatic symptoms.

The two cases of recovery are as follows:

Case XXXVIII (Delaire[4]).—A woman of thirty-four, when convalescent from typhoid fever, was suddenly seized with pain in the hepatic region, followed by enlargement of the liver below the level of the umbilicus by the twentieth day. Two days later she was seized with sudden thoracic pain, and was nearly strangled by an immense quantity of pus which she expectorated, an abscess of the liver having evidently ruptured through the diaphragm and discharged through the lung. She recovered her health entirely, when a year later a renewed attack of the hepatic abscess occurred, and ruptured at a spot near the right iliac spine after the use of Vienna paste. Finally, however, the abscess had to be opened, and she recovered.

[1] Lancet, 1874, ii, 552. [2] Deutsch. Chir. Lief, 45 c. Erste Hälfte, 238.
[3] *Loc. cit.* [4] Gaz. des Hôp., 1869, No. 111, 437.

Case XXXIX.—Sidlo[1] reports the following case: A girl ten years old became ill with typhoid fever at the end of February, 1873. By the fifteenth day defervescence had occurred. On the seventeenth day, however, she complained again of headache, and had marked chills, followed by delirium, enlargement of the spleen, and meteorism. On the thirty-second day she first noticed pain in the hepatic region. Three days later there was marked swelling, with jaundice. On the forty-fourth day there developed also fluctuation, first over the left and then over the right mastoid, and in the right temple. After these abscesses had run their course, she complained of pain in the region of the fifth and sixth ribs, followed by swelling. The whole axilla in a short time became a monstrous abscess, and she lost flesh to such a degree that she was little more than a skeleton. On the eightieth day the tumor in the axilla began to shrink, and on the eighty-fourth, after severe pain in the abdomen, she began to pass blood and pus by the bowel. In the afternoon of that day she had 25, and in the night 10 additional, movements. On the next day there were 20 other movements, during which scybalous masses and pus were passed. With this her liver dulness and the jaundice diminished, and on the one hundred and twentieth day she was entirely well.

Surgery can do but little actively in these cases. The treatment must be entirely symptomatic. The few that recover will be most fortunate, and owe quite as much to nature as to the surgeon.

Suppurative pylephlebitis occasionally, but very rarely, follows typhoid, and is sometimes the cause of abscesses. It is probably a result of thrombosis of the vena portæ. Bückling[2] found throm-

[1] Der Militär Arzt, Wien, 1875, No 23, p. 20.

[2] Fälle v. Leber Abscesse, Berlin, 1868.

bosis of the vena portæ in two cases. Such thrombi would readily become infected by any of the bacteria above mentioned.

The pylephlebitis may result in abscesses proper in the liver, as in Romberg's case,[1] in which the thrombosis extended from the ileo-colic veins to the portal vein and its branches in the liver. He refers to four other cases. Staphylococci were found both in the thrombi and in the abscesses.

Osler, in his extensive pathological experience, has seen but a single case.[2] In this case multiple abscesses occurred in the mesentery, which "fluctuated like a sac of pus." "Outside the liver the portal vein was represented by an elongated abscess with thick, irregular walls." The splenic vein also was closed by a thrombus.

In Lannois' case,[3] a man of thirty-eight died three days after admission with typhoid pleurisy and supposed tubercular peritonitis. The necropsy showed adherent ante-mortem clots in the portal, splenic, and inferior mesenteric veins, with numerous abscesses in the liver, in which was found the typhoid bacillus associated with other organisms.

In these cases, also, surgery can offer no help. They are necessarily fatal.

II. Typhoid Affections of the Gall-bladder.—In striking contrast to the rarity of surgical complications and sequels of typhoid in the liver is their relative frequency in the gall-bladder. As this subject is of great importance, and is comparatively new, I shall consider it somewhat fully. Dr. Westcott

[illegible] [2] Trans. Assoc. Amer. Phys. 1[illegible] x, 352.
[3] Rev. de Méd., 1885, 406.

has tabulated 74 cases of typhoid infection of the gall-bladder accompanying or following typhoid fever. Of these, 30 resulted in perforation.

Five important papers have been published lately in connection with this complication. Curiously enough, two of them were presented at the same meeting of the Association of American Physicians, in May, 1897, and are to be found in Volume XII of their transactions. One is by A. Lawrence Mason, on "Gall-bladder Infections in Typhoid Fever" (p. 23), the other on "Hepatic Complications of Typhoid Fever," by Prof. Osler (p. 378), in which he includes affections of the gall-bladder.

The most important papers preceding these two are one by Chiari,[1] and another presented by him to the International Medical Congress in Rome, in 1894. Another important publication is that of Dupré.[2] Murchison and a number of other writers referred to suppurative or catarrhal inflammation of the gall-bladder in the same year in which my Toner Lecture was published (1876). Hagenmüller[3] collected 18 cases.

As in so many other surgical relations of typhoid fever, the discovery of the bacillus of typhoid has thrown an entirely new light on both the etiology and pathology of the disease. Gilbert and Girode[4] first demonstrated the presence of the typhoid bacillus in a suppurative cholecystitis in 1890, and followed

[1] Ueber Cholecystitis Typhosa, Prager med. Wochen., 1893, No. 22.

[2] Les Infections Biliaires, Thèse de Paris, 1891.

[3] Cholecystitis Typhosa, Thèse de Paris, 1876.

[4] Contribution à l Étude Bactériologique des Voies Biliaires. Mém. de la Société de Biologie, 1890, La Semaine Méd., 1890, No. 58.

up the subject in a second paper in the same journal[1] in 1893. The bacillus of typhoid has been found not only during or immediately after the fever, but also at periods long after the fever. Thus, Dupré[2] relates the case of a woman of forty-five, who died after the operation of cholecystotomy. The typhoid bacilli were found in her gall-bladder six months after the fever, and Chantemesse,[3] in a similar case, found them eight months after the fever. Von Dungern[4] reports the following very remarkable case, in which, after several recurring attacks of different disorders, presumably nearly all due to the typhoid bacillus, an abscess finally formed around the gall-bladder, possibly due to perforation. In the pus a pure culture of the typhoid bacillus was found fourteen and a half years after the fever, and the blood of the patient reacted promptly to the Widal serum-test.

Case XL.—A woman, age forty-six, was ill of typhoid fever in November, 1882, for four weeks. There were no symptoms in the region of the gall-bladder. In August, 1887, she suffered from a miscarriage, at the end of which she suffered from pains in the region of the heart, with severe vomiting, but without jaundice. In May, 1888, and in February, 1889, similar attacks came on, in the last of which she had some jaundice. A similar attack again occurred in June. She was then well until July, 1895, when she fell ill with gastralgia. In September, 1895, she had an attack of periostitis of the lower jaw, which got well after the discharge of a

[1] Cholecystite Purulente Provoquée par le Bacille d'Eberth, Mém. de la Société de Biologie, 1893, p. 956.

[2] *Loc. cit.* [3] Traité de Méd. i, 764.

[4] Münch. med. Wochen., 1897, No. 26, 699.

small sequestrum. On October 6, 1896, renewed attacks of colic began in the region of the gall-bladder. At the same time a firm tumor was discovered. By the middle of February her condition was still worse, with constant pain in the right hypochondrium and chills. The tumor in this region had now enlarged to the size of a child's head; it was scarcely movable; there was no jaundice; fluctuation was obscure; the tenderness not marked. The urine showed neither albumin nor biliary coloring matter. Kraske made an incision over the tumor and evacuated 150 c.c. of brownish yellow pus not colored with bile. Efforts to determine the relation of the abscess to the gall-bladder were naturally very limited, on account of the danger of breaking down the adhesions, but a sound penetrated ten cm. upward, backward, and inward in the direction of the gall-bladder. No stones were felt. The patient made an excellent recovery. A careful examination seems to have been made of the pus, which showed unquestionably the presence of the typhoid bacillus in pure culture. The blood of the patient reacted to the Widal test in concentration of 1 to 80.

It is greatly to be regretted, of course, that at the time of the necrosis of the jaw no bacteriological examination was made, but in view of the many cases already noted in this monograph, there can be no reasonable doubt, I think, that this was due to the typhoid infection. It would seem, also, that the repeated attacks of pain in the region of the gall-bladder, even though they were separated by years, are unquestionably to be attributed to the infection of the typhoid bacillus which was found at the time of the operation.

It is very remarkable, indeed, that after so long a

time as fourteen and a half years typhoid bacilli should be found in pure culture. I have not included the case in the table of cases of perforation of the gall-bladder, as it is not absolutely certain that it was such, though I think the probabilities would point distinctly in that direction.

Chiari reported 22 cases of typhoid, in all of which bacteriological examinations of the contents of the gall-bladder were made. In 19 of the 22 he found the typhoid bacillus, and he concluded that the presence of the typhoid bacillus in the gall-bladder was the rule. Councilman, in the discussion on Mason's paper, says: "I have come to regard the gall-bladder as one of the surest places to obtain a pure culture of the organism." Welch and Blackstein[1] were among the earliest to demonstrate the presence of the typhoid bacillus experimentally in the bile of a rabbit as long as one hundred and twenty-eight days after recovery from the inoculation.

This almost constant infection of the gall-bladder led Chiari to suggest that very possibly it bore a causative relation to relapses in typhoid in this way. When the patient is sufficiently convalescent to be allowed a more generous diet, an increase in the quantity of food taken may arouse the liver into greater activity than heretofore, and the increased flow of bile may flood the system with numerous bacilli of typhoid, which may give rise to a reinfection causing the relapse. One would suppose that the condition of the blood-serum would be a protection against the new invasion, but in certain persons this seems to fail.

[1] Johns Hopkins Hospital Bull., Aug. 19, 1891, p. 121.

While it is perfectly true, as already indicated in a number of cases, that Eberth's bacillus may be present, there are often no apparent pathological changes caused by its presence. It is noticeable, also, that Osler states that in the histories of the seven fatal cases in which the typhoid bacilli were found in the gall-bladder the infection was so latent that there were no hepatic symptoms observed during life.

Not only may the bacilli be present in the pus, but also in the walls of the gall-bladder, causing local necrosis. Thus, Chiari[1] reports such a case, in which there were several necrotic patches on the walls, and the patient died from peritonitis, the direct result of the cholecystitis.

Milian[2] found the typhoid bacillus itself in pure culture in gall-stones and in the wall of the gall-bladder. The case was a woman, twenty-four years of age, who died on the sixteenth day after what he calls "hypertoxic" typhoid fever. She had never had either hepatic colic or other symptoms of gall-stones, but 25 were found at the post-mortem. In this particular case he believes that the gall-stones resulted from the typhoid bacilli, but it seems doubtful whether within sixteen days 25 gall-stones could have been so formed.

Prof. Welch also informs me that he has frequently found the colon bacillus not only in the bile, but also in gall-stones themselves.

Fournier,[3] in 100 cases of gall-stones removed at necropsies, found living or dead bacteria in the gall-

[1] *Loc. cit.* [2] Gaz. Hebd., Nov. 26, 1896, 1137.
[3] Origine Microbienne de la Lithiaise Biliaire, Thèse de Paris, 1896.

stones in 38 cases. The colon bacillus was the most frequently found, the typhoid bacillus being next in frequency. He believes that an angio-cholitis results either from a stasis of the bile or from its infection by the typhoid bacilli. This is followed by deposition of the salts of the bile in the form of gall-stones. Possibly, even the mere presence of the bacilli may be provocative of the formation of gall-stones. Thus, Gilbert and Domenicini[1] first positively identified the bacilli as the nucleus of gall-stones, and Hanot[2] confirmed this observation.

The facts above stated make it perfectly clear, therefore, not only that the bacilli do reach the gall-bladder, and may be found in the contents of the gall-bladder, and even in gall-stones, but that they are sometimes found in its walls, and may there induce local necrosis. Not seldom, also, they probably are the direct cause of gall-stones and the resulting biliary attacks.

Besides the bacilli of typhoid, other bacteria are frequently found in the gall-bladder. Létienne,[3] out of 42 cases, found bacteria present in 24. These were most commonly the staphylococcus and the colon bacillus.

Flexner[4] has stated that of 14 cases of typhoid in which the contents of the gall-bladder were examined, typhoid bacilli were found seven times, the bacillus coli communis three times, the streptococcus pyogenes once, and the proteus vulgaris once.

The question now arises, By what route do the bacilli reach the gall-bladder? Naturally, two direct

[1] Soc. de Biol., June 16, 1894.
[2] Bull. Méd., Jan. 22, 1896.
[3] Arch. de Méd. Expérim., 1891.
[4] Osler, *loc. cit.*, p. 385.

modes of infection are possible, and in all probability either or both may be assumed to be the route of the infection. First, the actual finding of the bacilli in the blood itself by a number of observers (p. 58) suggests at once the possibility of infection through the circulation, and this, in Councilman's opinion, is the common method of infection. In addition to this, as the bile ducts are freely open into the intestinal canal, there would seem to be no reason why the infection might not be a direct retrograde one through their continuity.

Through the courtesy of Dr. Mark W. Richardson, of Boston, I am able to give the results of his as yet unpublished investigation bearing upon this point:

"In three recent typhoid autopsies, cultivations from the colon, ileum, jejunum, duodenum, and gall-bladder were made, to see if a direct chain of bacilli, so to speak, could not be traced from intestine to gall-bladder.

"In Case I, though the case was clinically typhoid, and, moreover, gave a marked typhoid serum-reaction, not a typhoid bacillus could be found in any organ.

"In Case II there were abundant typhoid bacilli in the gall-bladder, but none could be obtained from the intestine.

"In Case III we found pure cultures of the typhoid bacillus in gall-bladder, duodenum, and jejunum, and it was only when the ileum was reached that colon bacilli began to appear at all.

"This result is quite remarkable, I think, and would seem to strengthen the theory of an ascending infec-

tion through the gall-ducts, though, of course, it is perfectly conceivable that the bacilli in the duodenum were *excreted* from a gall-bladder which was infected originally through the blood."

A second question requiring answer is whether the typhoid bacilli are capable of producing cholecystitis, empyema of the gall-bladder, and ulceration independently of gall-stones. Of this I think there can be no doubt, especially in view of the facts above stated, and of the suppuration and ulceration which nearly every chapter in this book shows to be caused by them. Of the 74 cases of biliary infection in our table, in 18 cases gall-stones were found. In a few others, which did not come to a post-mortem, there may have been also gall-stones present. In 38 cases, however, no gall-stones were present, and of these 8 were in persons under twenty-five years of age and 6 under fifteen, at which period of life it is extremely uncommon to find gall-stones. In 34 of the cases reported, cholecystitis, empyema, or ulceration were found in the gall-bladder without any gall-stones, and the typhoid bacilli were identified by bacteriological examination in 11 cases.

Not only were the bacilli present, but in several cases, some of which have been alluded to, there were present such ulcers as very soon would have led to perforation if life had been somewhat more prolonged.

Another interesting question already referred to is whether the typhoid bacilli of themselves give rise to gall-stones by producing stagnation of the bile, as has been suggested by Bernheim.[1] Certain it is that occa-

[1] Dict. Encyclopédique de Dechambre, 1889, Article Ictère.

sionally biliary colic occurs either during or after typhoid and suggests this possibility, as in the case of Chantemesse with a slow convalescence in which, after six months, an operation for gall-stones was done and a pure culture of typhoid bacilli was found in the gall-bladder.

The most important article dealing with this aspect of the subject is the paper of Dufourt.[1] He refers to 19 patients with gall-stones whose first attack followed typhoid fever in varying periods: in the second month in two cases; the third month in six; the fourth, in three; the fifth, in one; and in the other five as long as ten months or later after recovery. In Osler's first case, in convalescence in the fifth week after the incidence of the fever, the patient, who had never had hepatic colic, had an attack of sudden and severe pain in the right hypochondrium. This was followed by four other attacks, and though an operation did not reveal a gall-stone, one was discharged nearly two months after her last attack of biliary colic and nine months after her fever. This case will be referred to again under the heading of perforation.

The fact also that Dr. Welch has found the colon bacillus and Milian and Fournier the typhoid bacillus in gall-stones themselves, suggests the probability that either or both of these bacteria may bear an etiological relation to the gall-stones.

It is but reasonable to suppose that a gall-bladder already irritated and, it may be, inflamed by the presence of gall-stones, would be more likely to suffer from any invasion of the typhoid bacillus, and

[1] Rev. de Méd., 1893.

the very fact that in 18 cases out of the 74 of biliary infection gall-stones were found, makes it still more likely that gall-bladders containing such calculi readily fall victims to the ravages of the disease.

Surgically speaking, biliary infections may be divided into two clinical forms: First, those in which cholecystitis and empyema exist with or without gall-stones, and, secondly, a very much more important class, those in which perforation of the gall-bladder takes place.

The *symptoms* in the first class of cases may be remarkable by their utter absence (*cf.* Osler, *supra*), so much so that in at least one-half of the cases of biliary complications the fact is wholly unsuspected. This is partly on account of the latency of the symptoms, but also largely on account of the stupor of the patient. In these cases the cholecystitis and gall-stones are only discovered at the post-mortem. Ordinarily, if any symptoms are observed they will be, pain in the region of the gall-bladder, and the distended gall-bladder will be discovered both by touch and on percussion. The tenderness, as pointed out by Mayo Robson,[1] is usually at the junction of the upper two-thirds with the lower third of a line drawn from the ninth rib to the umbilicus.

As a rule, no surgical treatment can be instituted, but Mason's first case is a striking illustration of the occasional value of surgical interference. In brief, this is as follows:

Case VII.—A woman, age thirty, toward the end of the third week complained of pain in the

[illegible] Med. Jour., March 11, 1899.

right hypochondrium. A tumor four inches in diameter developed between the costal border and the umbilicus. The tumor was very painful, dull on percussion, and moved with respiration. A week earlier, when she was admitted, the liver dulness had been normal, and there had been no pain in this region. Rupture of the gall-bladder being deemed to be imminent, Mason at once tapped it at a point an inch below the costal margin and 4½ inches from the umbilicus. Three and a half ounces of sero-purulent fluid were withdrawn, looking more like urine than bile. No gall-stones were felt. The relief of the pain was immediate, all the urgent symptoms disappeared, and the patient made an excellent recovery. The typhoid bacilli were found in the fluid withdrawn. Pure cultures prepared from the fluid reacted to Widal's test.

It is not often that such simple treatment will be followed by so happy a result, but it certainly should be employed in all cases of similar distention of the gall-bladder.

The following case, treated on the conservative plan then in vogue, also happily recovered:

Case XLII.—Salzmann[1] reports the case of a woman, age forty-two, who, in the autumn of 1865, fell ill of a severe attack of typhoid fever, which involved five members of the same family. During the height of her illness a tumor was suddenly perceived in the abdomen between the umbilicus and the liver. This must have formed suddenly, as repeated prior examinations had not revealed it. When found it was the size of an orange. Its appearance was followed very soon by an intense jaundice, which, however, gradually subsided, and

[1] Med. Correspondenzbl. Württemburg. Arztlich. Vereins, 1870, xl, 84.

she recovered from the typhoid fever. The tumor, however, did not disappear. In December, 1866, a year after the illness, the tumor broke through the abdominal wall and gave exit to a considerable amount of clear serum. For a year after this the tumor gradually shrank, but left a continually discharging sinus. In 1868, two and a half years after the onset of the trouble, the discharge from the sinus increased considerably. Toward the end of 1868 her mental condition became very bad, so that insanity was feared. On January 31, 1869, over three years after the beginning of the disease, a gall-stone, which was distinctly perceived in the fistula, was removed; the patient made a good recovery, and five months later the fistula finally closed. Her mental condition, however, did not improve very much.

The next case, also of operation on a distended gall bladder, I owe to Professor Osler:

Case V. (Osler).—K. T., colored girl, age [illegible], admitted to the medical department of the Johns Hopkins Hospital September 15, 1897. [illegible] for about three weeks. Twenty-[illegible] she had 15 stools; tem-[illegible]

[illegible]; was noted that the con-[illegible] She had complained [illegible] great sensi-[illegible] the abdomen [illegible] distinct mass. [illegible] the evening [illegible] sensitive [illegible] three cm. [illegible] mass [illegible] two cm. [illegible] made

"On September 20th there was still so much tenderness that it was decided to operate, which was done by Dr. Cushing. The gall-bladder was distended and very tense, and 120 c.c. of fluid were aspirated. A drainage-tube was inserted and the wound partially closed and packed with gauze.

"The patient's condition was very bad from the day after admission. The temperature had been persistently above 105°, and on the 16th reached 106.4° and at the time of the tapping of the gall-bladder was 106°. The patient died just twenty-four hours after the operation.

"The autopsy by Dr. Livingood showed characteristic typhoid lesions in the intestines. The gall-bladder contained some clotted blood; no gall-stones. Typhoid bacilli were obtained in pure culture from the fluid removed by aspiration, from that removed at operation, and at autopsy."

The following case of doubtful etiology is of especial interest both surgically and bacteriologically:

Case XLIV.—Mark W. Richardson[1] reports the case of an elderly woman who suffered for nearly four weeks with fever, nausea, and considerable pain and tenderness in the right iliac region. This was thought at first to be due to appendicitis. No rose-spots were seen. Later there was some cough and rusty sputum. She had a "typhoid look." There was no enlargement of the spleen. After about two weeks a firm, rounded tumor was found at about the level of the iliac spine. It was tender, painful, and movable and extended around to the loin. The urine was negative. No positive diagnosis was made, but it was thought, possibly, to be a pyo- or a hydro-nephrosis. About April 1st an

[1] Boston Med. and Surg. Jour., Dec. 16, 1897.

incision was made in the back by Dr. Maurice H. Richardson and disclosed a normal kidney. This incision was closed and another was made over the gall-bladder, which was found to be excessively distended. Over half a pint of dirty brownish fluid was evacuated. A most careful examination showed a pure culture of typhoid bacilli. The blood, a week after operation, gave no serum reaction, nor did the feces at the same time show any typhoid bacilli. A gall-stone impacted in the cystic duct was removed. The patient, when the case was reported, was recovering without complications.

As to whether this was a case of typhoid fever followed by cholecystitis due to a typhoid infection of the gall-bladder, or possibly analogous to the case reported by Osler (see p. 48) of typhoid infection of the gall-bladder without typhoid fever, the fortunate recovery of the patient prevents a positive opinion.

Perforation of the Gall-bladder.—Much more important surgically are the cases of perforation of the gall-bladder; there are 30 of these in our table. Of these 30, four have been operated upon. This opens a new field in the surgery of typhoid fever which is of great importance and demands most careful study. The facts already stated as to the infection of the gall-bladder, of the presence of gall-stones, of the presence of ulcers in the walls of the gall-bladder, and of necrotic areas in the wall, suggest the probability, and also the ways, in which perforation may occur.

The *symptoms* will be those usual in perforative peritonitis from any cause, such as perforation of the appendix, of the stomach, or of a duodenal ulcer. Sudden and severe pain is, of course, the most

prominent symptom. This pain will be most intense in the region of the gall-bladder. If the gall-bladder is much distended and perforation takes place toward the fundus, the seat of the most severe pain may be nearer to the umbilicus or in the right iliac fossa. In such cases it is not at all improbable that it would be very difficult to differentiate it from the perforation of the bowel from typhoid ulcer or perforation of the appendix. The difficulty of making a differential diagnosis is of less importance than might appear at first sight, since in the present state of the surgery of typhoid fever the same treatment should be unquestionably instituted. Not only will there be severe pain, but collapse may set in very quickly. If the collapse be primary, partial reaction may follow. The abdomen will become distended and, unless the surgeon interferes, death will soon follow, with all the ordinary signs of perforative peritonitis.

Of the 30 cases of perforation reported in our tables, 3 recovered and 27 died.

All of those that recovered were operated upon.

Sex.—There were: males, 11; females, 11, in 22 cases in which the sex is stated.

Age.	Recovered.	Died.
Under fifteen, ... 9	1	8
Fifteen to twenty-five, . 5	0	5
Over twenty-five, . . . 9	2	7

Date of onset:

During the first week, ...	1
During the second week, ...	3
During the third week or later, ...	19

In a small number of cases the exact time is not

stated, but is given as "during the course" of the fever. This I have assumed to be in the third week or later.

Of the cases in which perforation took place, 7 had gall-stones and in 16 no gall-stones were found.

Of 26 cases not operated on, every one died.

Of 4 cases that were operated on, 3 recovered and 1 died.

The great mortality after perforation of the gall-bladder, without operation, and the fact that three cases out of four operated on recovered, is our strongest argument and best incentive to surgical interference in similar cases.

As the four cases operated on are so important from the surgical point of view, I append a brief résumé of each.

Operation.—The operation for peritonitis following perforation of the gall-bladder differs in no material respect from the operation for perforation of the bowel, and I will refer the reader, therefore, to Chapter XV for the principal points. Two others need some consideration.

First, the incision. The best line of incision is over the tumor, if there be one; if not, then at the outer border of the right rectus muscle. In case the gall-bladder is distended toward the median line, a median incision may be best. In three of the four cases operated on the incisions were respectively: in the median line; parallel with the ribs; and the incision for appendicitis: the exact incision in the fourth case is not stated.

The only other peculiarity, perhaps, that I need mention is the method of dealing with the gall-bladder

itself. If the walls of the gall-bladder in the neighborhood of the perforation allow of it, one or two rows of Lembert sutures, inverting the peritoneal coat, is the best method of repairing the perforation. Should this not be possible, then the best plan would be circular packing around the opening with iodoform gauze, so as to form a defensive wall for the abdominal viscera, leaving in the center a sort of well leading down to the perforation. This should be separately packed. The incision should never be entirely closed, drainage being essential.

The *prognosis* in perforation of the gall-bladder, as distinguished from perforation of the bowel, is certainly very much more encouraging, if we may draw any inferences from so small a number of cases. This would naturally be the case, since the contents of the gall-bladder are less irritating than those of the intestine.

I append a résumé of each of the four cases of perforation of the gall-bladder which have been submitted to operation.

Case XLV (Williams and Sheild[1]).—"This carefully studied and admirably reported case should be read in full. A woman, thirty-one years of age, was first seen by Mr. Monier-Williams, September 18, 1894, her illness having begun on September 13th. The spleen was enlarged. There was some tenderness in the right iliac fossa, but no abdominal pain. September 23d, the eleventh day of the disease, she was seized early in the morning with sudden acute abdominal pain. Mr. Williams 'found her in a semi-collapsed condition, with feeble, fluttering pulse and cold, clammy skin, complaining

[1] Lancet, 1895, i, 534.

of pain in the abdomen. The temperature shortly before the commencement of the pain was 102.5°, but two hours later had fallen to 99°. Abdominal breathing was practically absent. The pain was diffused over the upper part of the abdomen, a spot a little above the umbilicus being, perhaps, the seat of its greatest intensity, and there was considerable tenderness over the region of the ascending colon. A possible perforation of the intestine was considered even at this early date. Opium was ordered and all food by the mouth stopped. Twelve hours after the collapse the temperature rose to 104.5°. In a few days all pain and tenderness had disappeared. By October 28th she was practically convalescent; the abdomen was natural in every respect. On the night of the 29th she had some slight abdominal pain, but not enough to keep her awake. On the morning of the 30th, however, she was suffering severe pain, which was referred to the region of the hepatic flexure of the colon, and there was considerable tenderness in this region; an area about the size of an orange became decidedly dull on percussion and very tender. Intestinal perforation was then again considered. The next morning, October 31st, the patient was much worse, with decided distention of the abdomen and signs of recurrent peritonitis in the right hypochondrium. On the 31st Mr. Sheild first saw her with Mr. Monier-Williams. Along with the symptoms already described, it is noted that 'the extremities were not cold, the tongue was clean, and the patient was rational and could converse. The face was pinched and anxious, but lacked the appearance of death-like collapse so noticeable in perforation of the intestine from typhoid ulceration. By the next day, November 1st, operation was determined upon, as the abdominal conditions were worse, though her general condition had improved.

A free incision was made in the median line above the umbilicus. The intestines were distended, red, congested, and covered with purulent lymph. Believing that a perforation existed toward the upper part of the intestinal canal, diligent search was made, but none could be detected, and while disentangling the adhesions and sponging away the soft lymph under the liver, the gall-bladder came into view, and its appearance was peculiar and suggestive. This viscus was deeply inflamed, of a dark plum color, rigid, thickened, and adherent, but not much enlarged, though tightly distended. The source of mischief was soon apparent, for low down near the neck of the gall-bladder was a sharply circular sloughing ulcer, the size of a three-penny piece. Its floor was bright yellow and it was surrounded by a vivid red zone of intense hyperemia. On stroking it with the probe, fluid escaped at one point, showing that leakage of the contents had already occurred. The gall-bladder was opened at its fundus, evacuating f℥iss of thick offensive pus not mixed with bile. Careful search for a calculus was made, but none could be detected. The sloughing ulcer near the neck of the gall-bladder had now quite given way, and I [Mr. Sheild] tried to unite it after the Lembert method, but the stitches cut their way out persistently, though deeply placed. It was obvious that the gall-bladder was too soft and lacerable to deal with, still less to bring it to the surface of the abdominal wound. The wound in the fundus was, therefore, attached to the parietal peritoneum and the parts under the liver cleansed by flushing with warm water and repeated sponging. A second incision was made at right angles to the former, reaching toward the right lower ribs to allow of more drainage. The intestine was relieved of flatus by puncture; a glass drainage-tube was now inserted, and a long and liberal slip of carbol-

ized gauze packed firmly around it, so as to flatten the empty gall-bladder against the surface of the liver and shut off the intestine from contact. The rest of the abdominal wound was closed. The operation lasted an hour. The patient was fed by the rectum; a saline purge was given two days after the operation. On the sixth day a purulent discharge was observed from the tube; no bile escaped. After this the patient made a very excellent recovery.'"

Case XLVI (Alexieef[1]).—"A girl, age five, was taken, November 5th, with parotitis, which suppurated on the left side. By the 29th the spleen was enlarged. The patient passed through a severe attack of typhoid fever. 'During the second week in December the patient became very drowsy and was delirious; there was tenderness in the ileo-cecal region. The râles at the bases more numerous; considerable expectoration; the roseola diminished, but was still present. During the third week in December the patient became markedly worse, complaining of pain, especially in the right side of the abdomen. When lying on her back there was to be made out, four or five fingers' breadth below the costal margin on the right side, a rounded tumor about half the size of a small hen's egg. On percussion the tumor was pear-shaped, and the dulness was continuous with that of the liver. There was marked motion with respiration; it was elastic and somewhat tender on palpation. Distinct fremitus was found to be present on palpation between the 18th and 20th. Diarrhea. The urine by this time had begun to contain a trace of albumin. On the nights of the 20th and 22d the patient became much worse, complaining of great abdominal pain. Finally she became greatly excited; jumped out of bed; did not

[1] Jour. Dietskaya Meditzina, 1896, No. 4, in the Amer. Jour. of the Med. Sciences, Oct., 1897, p. 460.

recognize those about her; and became very feeble and collapsed. Temperature, 36.6° C.; pulse uncountable. Tinct. valer. æth. was given hypodermatically; by the following morning she became somewhat calmer. At this time the tumor, which had been palpable the day before, was no longer to be made out. The abdomen was much distended and tender. The bowels moved only by enema.

"'On the 25th patient was worse, complaining of severe pain in the right side; abdomen much distended; everywhere tender. Cries out with pain. There is no jaundice. From the 29th to the 31st the temperature was somewhat lower, but the patient seemed much worse; lies on her back or on the right side, with the knees drawn up; can take no other position. The abdomen, especially on the right side, is distended and very tender. The outlines of the tumor, which were formerly made out, are not to be distinguished.

"'A diagnosis of cholecystitis or suppuration in the neighborhood of the gall-bladder was made, and operation advised. This was performed on the 31st, an incision nine cm. long, parallel to and two fingers below the costal margin on the right side, being made. On opening the abdominal cavity a serous fluid, colored with bile, escaped. The intestines were adherent; there was pus in the gall-bladder and surrounding it, suppuration having extended also into the pregastric area. The pus was emptied, drainage established, and the cavity packed with iodoform gauze. After the operation the patient was excessively weak, and 20 c.c. of salt solution were introduced hypodermatically.' The patient made an excellent recovery. On February 9th, however, a relapse occurred, from which she recovered by the 21st. During all this time the discharge in the healing wound had gone on satisfactorily. The discharge consisted of bile, mixed at times with

a little pus. The discharge stopped by the 24th and the spleen was no longer palpable. A slight attack of pneumonia occurred from March 13th to 17th, but by March 26th she was entirely well. Careful cultures from the pus showed pure typhoid bacilli.

Case XLVII (Osler[1]).—"A woman, age twenty-nine, was admitted to the Johns Hopkins Hospital, April 22, 1895, on the fourth or fifth day of typhoid. She passed through a period of pyrexia of twenty-seven days' duration. 'On May 22d she had an attack of sudden severe pain in the right hypochondriac region. The temperature rose to 102°, without a chill, and she had fever for thirty-six hours. There was no jaundice. Some time after she left the hospital, on June 21st, she had a second severe attack of pain in the same region, of great intensity. It was also not followed by jaundice. During the summer she had two other attacks, each lasting about eighteen hours, but without jaundice. She apparently had recovered entirely, when at noon on December 23d she vomited, and later in the day had an attack of very sharp and severe pain in the right iliac fossa. The pain came on in paroxysms lasting for a minute or more. The abdomen was flat, but over the entire right side there was muscular rigidity and a great deal of tenderness. The pain was localized in a small area close to McBurney's point. Any palpation in that region was very tender. Pressure on the left side of the abdomen caused a dragging sensation and pain between the navel and the anterior superior spine. The next day she had recurring attacks of pain over the whole right side.' A diagnosis was made by both Osler and Halsted of appendicitis. She became so much worse that at six o'clock the same evening Dr. Halsted operated. 'An incision was made for appendicitis. There were a few old

[1] Trans. Assoc. American Physicians, 1895, x, 388.

bands of adhesion seen about the cecum, but the appendix was not found, and it was evident that the seat of the trouble was not here. Just above the cecum the omentum was seen, adherent to the ascending and the hepatic flexures of the colon. In it was a small opening from which a thin yellow material flowed. At first this was thought to be perforation of the intestine in an old typhoid ulcer, but on enlarging the incision the opening was found in the gall-bladder, to which the omentum was adherent, and in the adhesions between the liver and colon there were several small pockets containing purulent yellowish material. The gall-bladder was enlarged and very tense; 100 c.c. of a clear fluid were removed, after which about 30 c.c. of thick purulent matter flowed out. The cystic and common ducts were explored, and it was thought that a stone was felt in the cystic duct, but on account of the depth of the duct and the numerous adhesions, and the difficulty of preventing infection of the peritoneal cavity, the gall-bladder was packed with gauze and the opening about it closed.

"'The patient did very well. On February 1st, upon introducing a probe into the sinus, a stone was felt, which gradually came out. She was discharged February 19th, and has remained well ever since.'"

Case XLVIII (Osler[1]).—"November 5, 1894, he saw a woman, age thirty-seven, at the end of the third week of a very severe attack of typhoid. The attack was very protracted and she was in bed nearly ten weeks. On January 1, 1895, she had been attacked suddenly with severe pain in the epigastrium, passing round the right side to the back. She had no chill. On the fifth day after the onset she became jaundiced. On January 21st she had a severe chill; on the 27th the temperature rose to nearly 105°, and she had nausea and vomiting. The abdo-

[1] *Loc. cit.*, p. 390.

men was distended and so tender that it was impossible to make a satisfactory examination. Dr. Halsted performed laparotomy on the 29th.

"'The surgical note states that on opening the abdomen the liver was seen to be enlarged, the gall-bladder projected below the right margin, and on its anterior wall a rupture was seen through which bile and purulent matter were oozing. The wall of the gall-bladder in the neighborhood was quite necrotic. The gall-bladder was incised, drained, and packed.

"'Cultures were made from the contents of the gall-bladder, and the colon bacillus was found. The colonies were worked over with the greatest care with reference to the differentiation of the typhoid from the colon bacillus.

"'The temperature fell after the operation and became normal on February 1st. She did very well for ten or twelve days; then she began to have nausea and vomiting, with marked reduction in the amount of the urine. There was no change in the jaundice, and she sank and died on February 21st.'"

TABLE II.—CASES OF OPERATION FOR PERFORATION OF THE GALL-BLADDER AFTER TYPHOID FEVER.

Reporter.	Reference.	Age.	Sex.	Location.	Date of Occurrence.	Operation.	Interval before Operation.	Result.	Remarks.
[1] Williams and Sheild.	Lancet, 1895, i, 534.	31	F.	Near base of gall-bladder.	Eleventh day.	Suppuration in gall-bladder. Intestines punctured to relieve flatus. Drainage and packing.	. . .	R.	See original paper for exact and valuable report, and page 265 for a full résumé.
[2] Alexieef.	Jour. Dietskaya Meditzina, in Am. Jour. Med. Sci., Oct., 1897, p. 466.	5	F.	Perforation of gall-bladder.	About fifth week.	Laparotomy. Packed and drained.	Uncertain; probably 2 days.	R.	Tumor in region of gall-bladder. Symptoms of perforation. Cholecystitis and empyema found at operation. No stones. Bacilli of Eberth in pus. Relapse in 12th week. Permanent recovery without fistula.
[3] Osler and Halsted.	Trans. Assoc. Amer. Phys., 1897, xii, 388.	29	F.	Perforation of gall-bladder.	Eight months after the fever.	Operation for appendicitis. Omentum adherent to colon. Several small abscesses between liver and colon. One hundred c.c. clear fluid and 30 c.c. pus evacuated from gall-bladder. Stone possibly felt and spontaneously discharged 6 weeks later.	18 hours.	R.	She had 5 attacks of pain in the region of the gall-bladder; the first in the 5th week of her typhoid fever.
[4] Osler and Halsted.	Trans. Assoc. Amer. Phys., 1897, xii, 388.	37	F.	Rupture of gall-bladder.	15 weeks after fever began.	Laparotomy. Liver enlarged. Gall-bladder ruptured; its walls necrotic. Contents purulent. Incision, drainage, and packing.	About 2 days.	D.	Only colon bacillus found in pus. Death on 23d day.

CHAPTER XVII.

TYPHOID AFFECTIONS OF THE SPLEEN.

AMONG the most constant places in which the typhoid bacilli are found are the bone-marrow and the spleen. On page 30 I have given a list of a number of authors who have found them in the spleen.

It was, therefore, rather a surprise to me that I was only able to cull from my second table of over 800 cases (there were none in my former series) only nine cases of abscess of the spleen; indeed, strictly speaking, only eight, for one of them was an abscess "about" the spleen. But as the bacillus of Eberth was found in it, and the spleen is so commonly invaded by the typhoid bacillus, it is reasonable to suppose that the spleen was at least its origin. There were also miliary abscesses in the kidney. In one case[1] the abscess ruptured, producing a fatal peritonitis. In two cases there was mitral disease, one[2] from an old rheumatism; the other[3] probably originated during the typhoid attack, as the bacilli of Eberth were found in the mitral vegetations. In this case there was also cerebral meningitis. Both of these cases may easily have been embolic in origin. One case[4] was complicated with gangrene of the

[1] Flavio, Gaz. degli Ospit., in Dublin Jour. Med. Sci., Nov., 1890, 445.
[2] Dunin, Univ. Med. Mag., Sept., 1895, 909.
[3] Vincent, Merc. Méd., Feb. 17, 1892, 73.
[4] Griesinger, Infect. Krankh., Virchow's Handb. Pathol., 1857.

lung, and another[1] with suppuration in the thyroid. One case arose "in the course" of the disease, four about the third week, and four as late sequels—three of them arising as late as the seventh and eighth week.

Every case proved fatal, as, in fact, would be expected. Three were only discovered at the necropsy, one (Flavio) having been overlooked in consequence of a left pleuro-pneumonia with effusion. If discovered and recognized, the only possible remedy would be a celiotomy—a desperate remedy; in most cases, probably, not even to be considered.

Six were males and three females.

The following case of leukemic spleen under my care seems to have resulted from typhoid:

Case XLIX.—Mrs. S. T., of Hamburg, Pa., was admitted to the Jefferson Hospital April 7, 1896, at the instance of Dr. Nice. She is a widow, age thirty-one. Both parents and six brothers and sisters are living and in good health. Three brothers and sisters died in infancy. Her menstruation began at fifteen, and has always been regular. She had a still-born child at eighteen, was married at twenty-seven, and has had one miscarriage since. Had influenza in March, 1893, when she was in bed for three weeks. In November, 1894, she had a severe attack of typhoid fever, confining her to bed for four months. During convalescence she complained of a severe, constant pain in the left lumbar region, with a point of great tenderness midway between the anterior superior spine and the costal cartilages of the tenth and eleventh ribs on the left side. She was unable to lie on the left side on account of the pain. She also had repeated irregular chills—some-

[1] Griesinger, *loc. cit.*

times one to three a day; occasionally a day or two passed without any. There were also profuse sweats, most frequent at night, but without any cough or expectoration. She has never been able to walk since her typhoid fever. Her abdomen began to enlarge soon after the fever, and has steadily increased in size to the present time. On admission her temperature was 102.4°, pulse 113, respiration 32. She looked very pale; her appetite

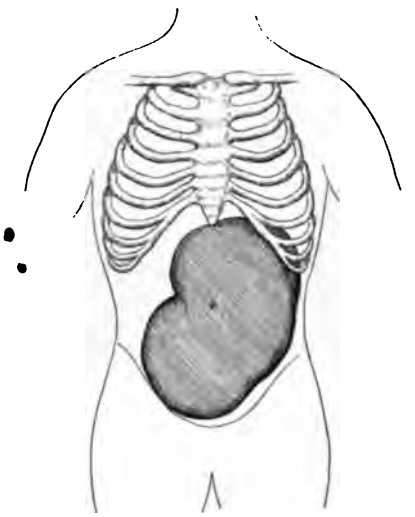

Fig. 4.—Area of tumor in Case XLIX.

was good; the abdomen was greatly distended by a large tumor, the area of which is marked in figure 4. It extended from the ribs to the brim of the pelvis, filled the entire left side of the abdomen, and reached to within an inch of the anterior superior spine of the ilium on the right side. About an inch above the level of the umbilicus was a notch on the right border of the tumor, and one at a lower level on the left border. Change of posture does not change the position of the tumor. Its right edge is

wedge-shaped. The surface of the tumor is smooth, regular, with firm resistance. The left costal cartilages are distinctly bulged forward by it, and there are no adhesions to the belly wall.

Dr. Kyle examined the blood and made the following report:

Hemoglobin, 45 per cent. of normal.
Red corpuscles, per cubic millimeter, total 2,720,000.
Red corpuscles, per cubic millimeter, normal, 2,500,000.
White corpuscles, per cubic millimeter, 336,000.
The appearance of the red corpuscles under the microscope was practically normal. A few were small (microcytes) and some slightly irregular in outline.
Of the leucocytes, many were granular and degenerating, some almost dividing. There were many large granular lymphocytes present. No malarial organisms. No typhoid bacilli.

As the case was one of leukemic spleen, I decided not to operate, and she was discharged April 17, 1896.

Dr. Nice writes me that she died on May 29, 1897, Nothing beyond the enlargement of the spleen was found at the post-mortem. Unfortunately, no opportunity was afforded for a bacteriological examination.

On page 163 I have given a résumé of a case of subdiaphragmatic abscess supposed to have arisen from the spleen.

CHAPTER XVIII.

TYPHOID AFFECTIONS OF THE SEXUAL ORGANS.

I. The Male Sexual Organs.—Very rarely the male genitals are attacked by gangrene (see p. 78). Janzion [1] has recorded a case of "malignant or ataxic fever" (typhoid?) in which priapism was the principal symptom. Andrew [2] has recorded a case of abscess of the prostate. One of my medical friends tells me that in convalescence from typhoid fever when a boy, he suffered from an attack of **urethritis** not due to the use of the catheter.

Case L.—Dr. A., at the age of fifteen, had an attack of typhoid fever followed by a relapse. No catheter was ever used. In the early part of convalescence a series of boils broke out over the body. A muco-purulent urethral discharge, with scalding following micturition, set in about the same time; there was no severe pain, no swelling of the meatus. The urethritis continued for about a week or ten days and then subsided spontaneously. Later in the convalescence the right testicle swelled somewhat and became a little tender. Though the swelling abated, this testicle has always remained somewhat larger than the other.

I only know of a single other published case of urethritis following typhoid fever [3]:

Case LI.—The patient was a soldier, who, after

[1] Ann. Soc. de Méd. de Montpellier, lv, i, 146. [2] Lancet, 1871, ii, 712.
[3] Legrain, Annales des Organes Génito-Urinaires, 1889, vii, 291.

typhoid, in the third week of convalescence, was attacked with urethritis. He had never had any venereal affection. For several days there was pain in urination, and later considerable discharge from the urethra, which became bloody. It was followed by cystitis of short duration. His recovery without treatment was complete at the end of three weeks. Bacteriological examination showed in the discharge numerous cultures of various micrococci, especially the staphylococcus pyogenes aureus, but no typhoid bacilli or gonococci. Legrain himself attributed this unusual urethritis to a possible necrosis of the urethral mucous membrane analogous to localized gangrene elsewhere.

The most frequent typhoid lesions are **orchitis** and **epididymitis.** It is somewhat strange that in the careful and extensive search through typhoid literature prior to 1876, I did not find a single case involving the testicle. In 1844, Velpeau,[1] however, I find, alluded to typhoid orchitis. Dr. Westcott has collected 32 cases, of which two had been reported prior to that date, but not found by myself.

The typhoid bacillus has been found in the testicle during typhoid fever, not only when it was not inflamed,[2] but has now been repeatedly found both in orchitis and epididymitis, and not only in the pus, but in the tissues. Gasser,[3] in a case of orchitis without suppuration, by an antiseptic puncture obtained a drop of fluid in which numerous typhoid bacilli were found. After recovery the testicle remained indurated. In Girode's case[4] of epididymitis, at the necropsy the suppuration was found to be intersti-

[1] Dict. En Trente Vol., Art. Testicule. [2] Dehu, *loc. cit.*, p. 82.
[3] Archiv de Méd. et de Pharm. Milit., 1895, No. 3, 228.
[4] Arch. Gén., 1892, clxix, 43.

tial and outside the canaliculi. A pure culture of the typhoid bacillus was found in the pus. The testicle was not involved. Though he was twenty-nine years of age, no spermatozoids were found, at least in the epididymis.

Tavel[1] reports a case of suppurative orchitis in which the typhoid bacillus was found in pure culture in the pus.

Ménétrier[2] reports a similar case. In both of these cases the epididymis escaped.

The date of onset, also, is a presumption that most of the cases were not due to a mixed infection by the pyogenic bacteria, but by the typhoid infection itself, for of 29 cases only a single one arose in the second week, seven in the third, and 21 occurred in convalescence—*i. e.*, after the third week. The marked tendency of almost all of the pure typhoid infections toward a late origin seems, therefore, to hold here also. One case was probably septic,[3] in which a parotitis arose on the fourteenth day and was followed six days later by orchitis, in a boy of four. It is not at all impossible, as Widal thinks,[4] that some of the cases may arise from thrombosis of the spermatic veins—an opinion, however, which was put forth prior to the discovery of the typhoid bacillus. Girode suggests that the typhoid bacilli gain access to the epididymis and the testicle from the urine. It is well known that the typhoid bacillus is often found in the urine (p. 32), and it is possible that this may be its

[1] Correspondenzbl. schweiz. Aerzte, 1887, 590.

[2] Pein, Thèse de Paris, 1890, 18.

[3] Bouchut, Mal. des Nouveaux Nés, 1867.

[4] Bull. Soc. Clinique, Paris, 1877, i, 142.

source, but the wide diffusion of infection through the blood (p. 23) seems to me to make it far more likely that the latter is its source. In Girode's own case the typhoid bacilli were not found in the seminal canaliculi (as would have been most probably the case had they come from the urinary passages) but in the inter-canalicular tissues.

The infection may be confined to the epididymis alone (4 cases), to the testicle alone (15 cases), or, as happened in 6 cases, both may be involved. The right side was involved 11 times, the left 9, and in one instance both sides.

Age.—

Under fifteen there were	2 cases.
From fifteen to twenty-five there were	11 "
Over twenty-five there were	10 "
	23 cases.

Date of Onset.—This occurred—

In the second week in	1 case.
" third week in	7 cases.
" fourth week in	3 "
" fifth and sixth weeks in	8 "
" seventh week and later	10 "
	29 cases.

The symptoms need no special description, except to call attention to the fact that the disease, especially in the epididymis, is often apyretic. Like lesions of the bones, where the infection is not mixed, but from the typhoid bacillus alone, the disease is local, and does not ordinarily, therefore, cause much constitutional disturbance.

The termination of the infection is in most cases

by resolution. This is expressly stated in 16 of the cases, and it probably followed in others. In the epididymis suppuration arose only once. When suppuration occurs in the testicle itself, its effects are apt to be more serious. This took place five times, and in three of them the sloughing was so extensive that practically the entire testicle was destroyed.

The mortality was small; only one case certainly died,[1] and in this death was the result not of the disease of the testicle, but of the lungs. In another the result is not stated;[2] but as it was probably septic, in a child of four, parotitis occurring on the fourteenth day and orchitis on the twentieth, it is doubtful whether so young a patient would survive the continued influence of the fever and these two early and probably septic complications.

II. The Female Sexual Organs.—In view of the occasional involvement of the testicle, it is strange that so few cases of pure typhoid abscess or other typhoid lesion of the ovary should have been recorded. Yet it has not wholly escaped the indirect malign influence of this upas-like fever.

Case LII.—Thus, Anger[3] has recorded the case of a woman, age thirty-three, who died, twenty hours after entrance into hospital, from typhoid of unstated duration. At the necropsy slight traction on an adhesion of a coil of intestine to the bladder at one of Peyer's patches tore a hole in the bladder at the site of a vesical ulcer. There were also abscesses in each broad ligament, due to a perforation of the rectum from ulceration.

[1] Girode, Arch. Gén., 1892, i, 43. [2] Bouchut, *vide ante*.

[3] Bull. Soc. Anat., 1865, 364.

Werth[1] describes the following case, in which an old dermoid cyst of the ovary suppurated eight months after the fever. Fortunately, a bacteriological examination was made, or the influence of the fever might well have been questioned. In the pus a pure culture of the typhoid bacillus was found. She recovered after operation.

Case LIII.—A woman, age twenty-nine, entered the hospital June 2, 1892, on account of an enlargement of the abdomen and pain in its right lower quadrant. The pain had suddenly seized her at the beginning of the year, while she was working, and had recurred at shorter or longer intervals ever since, later becoming more persistent and sometimes very severe. Soon after the beginning of the pain the patient observed that the abdomen was swollen, and in October and November, 1891,—that is to say, about eight months prior to her entering the hospital,—she had an attack of typhoid fever which, as was ascertained from her physician, was of moderate severity and without complications. On entering the hospital her abdomen was markedly distended by a tumor which plainly fluctuated. It was moderately movable and somewhat painful. The greatest circumference of the abdomen over the middle of the tumor was 78.5 cm. It rose 21 cm. above the symphysis. The uterus lay behind it. Operation June 4, 1892. The tumor was removed with some difficulty on account of adhesions, but the operation was finally successfully accomplished. The wall tore during removal, and some masses of fat, in which were a number of dark hairs, escaped. The tumor originated in the left ovary. The right ovary had a cyst as large as a lemon. This was opened with scissors and the wound was sutured. Bacterio-

[1] Deutsch. med. Wochen., 1893, No. 21.

logically the pus from the tumor showed a pure culture of the typhoid bacillus.

Case LIV.—Sudeck[1] reports the following similar case: A woman, age thirty-two, a widow for eleven years, suffered for several years with pain in the lower abdomen, dysuria, and constipation. Seven weeks before entering the hospital she fell ill with typhoid fever. Three weeks before her entrance into the hospital she noticed that the abdomen was swollen, and she had again pains in the abdomen and difficulty in passing water. On the 5th of November, 1895, when first seen, a marked tumor was found in the hypogastrium, extending into the pelvis, and to a little above the navel; fluctuation was uncertain. The uterus was anteflexed, lay somewhat to the left, and moved with the tumor. The cavity of the uterus was seven cm. long. Hectic fever set in and the diagnosis of a suppurating ovarian tumor was made. On the 11th of November Dr. Sick operated. The tumor was in the right ovary and was as large as the head of a ten-year-old child. It was removed without difficulty. The walls were about one cm. thick. The contents were a thick pus and fibrin with chocolate-colored fluid, smelling very badly. No bacteria could be found by the microscope. Cultures were made and the typhoid bacilli were recognized in pure culture. In the wall of the cyst were found some diplococci. The patient made an excellent recovery.

Case LV.—Pit'ha[2] reports a third case of suppuration in a dermoid cyst in which a pure culture of the typhoid bacillus was found. He attributes the suppuration entirely to the bacillus of Eberth. The patient was a woman twenty-five years of age. She was admitted to the hospital on February 6, 1897. Two years before he saw her she had suffered

[1] Münch. med. Wochen., 1896, No. 21, 498.

[2] Centralbl. f. Gynekologie, 1897, No. 37, p. 1109.

from chlorosis, during which time her menstruation ceased for a year and a half. Four months prior to her admission she fell ill with typhoid and was in bed for four weeks. In the fifth week she observed on the right side of the lower abdomen a painful tumor without definite limits. Gradually it became well delimited. Her menstruation had not returned since her sickness; appetite was poor; bowels irregular. She was evidently a poorly developed woman. In the lower half of the abdomen was a tumor which reached up to the navel, and, especially at the sides, showed fluctuation. It was not movable, and caused but little pain. A vaginal examination showed that the tumor pushed the posterior vaginal wall markedly forward, and the uterus was lifted and pressed against the symphysis, being covered by the bladder, which last reached half way to the navel. Examination by the rectum showed that the whole pelvis was filled with the tumor, that the recto-vaginal septum was pushed downward, and both the vaginal and the rectal wall arched. The rectal wall, however, was movable over the tumor. On the right side the tumor was connected with the bones of the pelvis by a hard, tender, solid mass. Otherwise the lower part of the tumor was elastic and fluctuating. Operation was done February 21, 1897. Some of the fluid was removed by a puncture and immediately examined microscopically. A great mass of polynucleated pus-cells in degenerative stages was found, but no micro-organisms. Accordingly, the tumor was opened through the posterior wall of the vagina by the thermo-cautery, evacuating three liters of pus, in which also were some hairs, making certain the diagnosis of a suppurating dermoid cyst. Examination with the finger showed that it was a multilocular cyst, and that vaginal extirpation would not be advisable, especially on account of its adhesions. It was, therefore, thoroughly washed out and the vagina

tamponed with iodoform gauze. Laparotomy was immediately done. The bladder reached a hand-breadth above the symphysis; the uterus was small; the dermoid cyst arose from the right ovary. There were some adhesions to the intestine, which were broken up by blunt dissection, but it was not possible to lift the tumor until the adhesions on the right side of the pelvis were broken up, which required considerable force. The pedicle was divided by the Paquelin cautery; the pelvis cleansed with sterile compresses; the raw surface of the pelvic adhesions were covered by a peritoneal flap from the right broad ligament. The left ovary was small. The opening in the vagina was closed with catgut and the abdomen entirely closed without drainage. Examination of the extirpated cyst showed that necrotic processes had begun at several points. Both bones and cartilage, as well as a considerable amount of black hair, were found in the tumor. The patient made an uninterrupted recovery. No micro-organisms whatever were found microscopically in the pus. The contents seemed to be chiefly disintegrated epithelial cells and polynucleated pus-cells. Cultures, however, were made which showed pure typhoid bacilli, both by morphological and biological tests and also by their reaction to Widal's serum test.

The conclusion of the author, therefore, was that the typhoid bacilli were the direct cause of the suppuration, and that they were present, as shown by the culture, four months after the fever.

Mabit[1] records a case of pyosalpinx, which, unfortunately, like that of Anger which occurred in 1865, has no bacteriological proof of its typhoid origin, and the remote date of its discovery and possibilities of intervening causes make it doubtful. A young

[1] Nouvelles Archives d'Obstét. et de la Gynécol., 1893, viii, 267.

woman of nineteen, when convalescent, developed recurring pains in the left side of the abdomen, with amenorrhea. She married at twenty-one, and at twenty-nine was cured of a left pyosalpinx by dilating and tamponing the uterus.

It is beyond the object of this monograph to consider the effect of typhoid upon pregnancy, but in passing it is not without interest to observe that Freund and Levy[1] report a case of spontaneous abortion at the fifth month, at the beginning of defervescence, the mother, a multigravida, having suffered from a mild attack. They found the typhoid bacillus in the blood of the placenta and in the spleen and the heart of the fetus, thus proving the direct transmission of the bacillus from the mother to the fetus, which could not have taken place by any other channel than by the blood. The child died immediately after birth, and the cultures were made twenty minutes later. No gross lesions were found in the fetus. Janizewski[2] reports a case of a woman, eight months pregnant, who, twelve days after her admission into the hospital with typhoid fever, aborted of a fetus which lived five days. Bacteriological examination of the fetus showed the typhoid bacilli in the spleen, intestines, mesenteric glands, kidneys, and lungs.

Similar cases of ante-natal infection of the fetus are reported by Neuhass, Chantemesse and Widal, and a number of others, to which the references are given on page 32.

Periuterine Hematocele.—In addition to these

[1] Berlin. klin. Wochen., 1895, No. 25.
[2] Presse Méd., March 24, 1894.

cases, Guyot[1] has collected seven cases of peri-uterine hematocele of typhoid origin, which, with another by Trousseau[2] originating, as he believed, in a sanguineous cyst of the ovary in a girl of sixteen, are the only cases Dr. Westcott has found. Whether, as Guyot thinks, they arose from an intestinal perforation is a question.

In Guyot's four personal cases the trouble began, in three cases, respectively in the fourth, sixth, and seventh weeks of the fever, and in the fourth at the beginning of convalescence. This last case died, unfortunately without autopsy; two of the other three recovered without operation, but with persistence of the tumor. In the fatal case pus was discharged both from the rectum and the bladder. No autopsy was allowed.

At the present day, undoubtedly, operation would be done in such cases, and as they all occurred practically in convalescence, the mortality would probably have been much less. The symptoms were in no wise different from the same disorder arising from other causes. No bacteriological examination was made, as it was before the discovery of the bacillus of Eberth.

Lesions of the vagina are chiefly caused by abscesses (see Chap. VI) or gangrene (see Chap. III), and result in simple perforations, vesico-vaginal and recto-vaginal fistulæ, destruction of more or less of the vagina, and sometimes entire closure of the vaginal outlet. These have already been considered.

[1] Etude sur l'hématocele périuterine survenant dans le cours on dans la convalescence de la fièvre typhoïde, Thèse de Paris, 1879.

[2] Clin. méd. Hôtel Dieu, 1865.

CHAPTER XIX.

SPECIFIC MIXED INFECTIONS.

TETANUS.—ERYSIPELAS.—ANTHRAX.—MALIGNANT EDEMA.

IN addition to the ordinary pyogenic infections giving rise to complications in the joints, bones, larynx, parotid gland, etc., and producing various forms of septic lesions, there are a number of cases of other mixed infections with certain specific micro-organisms to each of which a few words will be appropriate. Some of these specific infections may find a port of entry through the intestinal lesions, but most of them probably gain an entrance through the bedsores, furuncles, suppurating parotid glands, and other similar avenues in the skin and the mucous membrane of the mouth. Some peculiar and characteristic avenues of infection in the cases of tetanus are considered under that heading. Among these infections the most frequent is erysipelas; next to this is tetanus; and there are one case of anthrax infection and two of malignant edema. Before the discovery of the typhoid bacillus and the bacteria of these various infections their occurrence was inexplicable, but now this is perfectly plain. They are all cases of mixed infection with other specific germs in addition to that of typhoid.

Erysipelas.—The most important paper I have found in reference to erysipelas is Gérente's Th

de Paris, 1883.[1] He collected up to that date 64 cases of facial erysipelas in 3910 cases of typhoid fever, as is shown by the following table:

	Typhoid Fever.	Facial Erysipelas.
Chomel,	130	4
Louis,	134	3
Forget,	92	1
Jenner,	65	2
De Larroque,	105	4
Zuelzer,	84	3
Liebermeister,	1420	10
Zuccarini,	480	18
Griesinger,	500	10
Murchison,	900	9
Total,	3910	64

The references to the several papers may be found in the original thesis.

According to these figures, erysipelas is a complication once in every 61 cases of typhoid. Erysipelas, however, I believe is very frequently not mentioned, either because it is deemed an unimportant complication or because it is infrequent. That it is an infrequent complication is shown, I think, by the fact that the other cases in our table number only 29. There are a large number of cases of erysipelatous infection alluded to in a general way, but these would not serve my purpose for analysis. Only the few cases stated above were found described in such detail as to be useful.

The great majority do not arise in connection with the so-frequent bedsores, but more often, probably, from the fissures of the lips and other solutions

[1] L'Erysipèle de la Face dans le cours de la Fièvre Typhoide.

of continuity about the head and face. This accounts for the much greater frequency of erysipelas of the face than of other parts of the body. In addition to this, the erysipelas not infrequently attacks the mucous membrane of the mouth and pharynx, and may travel down the trachea and result in either bronchitis or pneumonia, or may involve the vocal chords, producing great hoarseness and also dysphagia. Of this Jenner has given two excellent illustrations,[1] the first of which, however, occurred in a case of typhus. In the skin, especially in such lax tissues as the eyelids, a local necrosis or gangrene may result. Meningitis does not seem to be a frequent sequence. The inflammation of the skin is sometimes stationary, or more commonly serpiginous or ambulant. Its duration may be fixed ordinarily at two weeks, but it varies very much. Two cases I have personally seen resulted in abscesses; one, an abscess of the scalp and eyelid, the other, over the mastoid process.

Case LVI.—A. C., age twenty-two, was admitted to St. Agnes' Hospital, April 14, 1891, on the fourth day of typhoid fever. She was under the care of Dr. H. A. Hare. On April 27th she developed facial erysipelas. By June 25th the erysipelas and typhoid had both subsided, but she was transferred to the surgical ward on account of an abscess over the left mastoid. On the next day I opened the abscess and packed it with bichlorid gauze. She was discharged, cured, July 4, 1891.

Case LVII.—J. W., age twenty-five, was admitted to St. Agnes' Hospital April 21, 1891, with begin-

[1] Med. Times (London), 1850, vol. xxi, p. 135; and Med. Times, n. s., vol. i (o. s., vol. xxii), 1850, p. 405.

ning typhoid fever, and placed in the medical ward. May 10, 1891, he developed facial erysipelas and the right eyelid threatened to slough. June 3, 1891, on account of an abscess on the back of his head, he was transferred to the surgical ward, the erysipelas having subsided. June 4, 1891, I opened the abscess. Another developed in the right eyebrow, and was opened June 8th. Both were healed by July 2, 1891, and he was discharged cured.

Unfortunately, no bacteriological examination could be made in either case.

Of the whole number, 82 were in the head and face, 1 of the foot and leg, 1 began in the arm, and 1, reported by Berthand[1] (in 1848, be it observed), was said to be "erysipelas of the iliac and renal veins." The autopsy showed a plastic and suppurative inflammation of these veins. Presumably this was a case of thrombosis.

One case[2] is remarkable for its malignant course. It broke out on the thirteenth day in a woman of twenty-five, who was four and a half months pregnant. Beginning in one arm, it spread over both arms, the chest, face, and head within twelve hours, when the patient died.

Nearly all the patients were between twenty and thirty—19 out of 28 cases in which the age is given. Five were below twenty (one each of twelve and eighteen and three of nineteen) and four were thirty-two, thirty-eight, forty, and forty-two years of age respectively. The two sexes were nearly equally attacked—16 males, 14 females. It begins, usually, either in the third week, when the fever is

[1] Gaz. des Hôp., v, 29. [2] Rosenberg, Columbus Med. Jour., 1890, 299.

at its height, or during convalescence, occasionally as late as the fifth to the eighth week.

Its fatality is not to be wondered at. Twenty-seven cases died and 32 recovered of the 59 cases in which the result is stated. This gives a mortality of almost 46 per cent. The later cases since the introduction of antiseptics give even a worse result, for of 23 cases of the 29 collected in addition to those of Gérente, 11 died and 12 recovered, a mortality of almost 48 per cent.

The only bacteriological examinations that have been made in these cases are by Rheiner,[1] and in his two cases the typhoid bacillus alone was found. Further examination will probably prove that the streptococcus is present, as a rule.

The *symptoms* are the usual ones of erysipelas, but instead of being aggravated from the combination with the typhoid fever, as one would suppose, they all seem to be ameliorated. There is, as a rule, less redness, less pain, and less swelling than in other forms of erysipelas, nor does the temperature, as a rule, rise so high as we would expect. Delirium also is not very marked. The cause of the great fatality seems to be rather the general condition of the patient, especially his double infection.

In the matter of *treatment* there is nothing which calls for special mention, except as to prophylaxis. The most scrupulous cleanliness, especially in any solutions of continuity,—such as fissures of the lips, nose, etc., bedsores, abscesses, furuncles, etc. Now that the great mortality from erysipelas is shown, a still more rigid antiseptic treatment, will,

[1] *Loc. cit.*

in all probability, avert the danger by preventing the secondary infection.

Tetanus.—In the Bibliography of my Toner Lecture I recorded six cases of tetanus, to which Dr. Westcott has added nine—15 in all.

The most interesting point in connection with these cases is the port of entrance of the infection. Thus, one case of the earlier series was believed to have arisen from a bedsore; one had a wound on his finger; one[1] probably developed infection through a blister; two[2] were attributed to a possible abrasion of hemorrhoids by a syringe (?); one[3] followed eight days after plugging of the nares for uncontrollable hemorrhage; one[4] followed the bite of a horse on the finger six weeks before; and one[5] occurred in a cavalryman who fell out of a window while delirious. In the earth where he fell the bacillus of tetanus was found. His occupation also is significant.

Of the 15 cases, 13 (including all of those of the second series) followed typhoid and two typhus; six were women, seven were men. Of the 15, ten died. Two women who recovered had menstrual irregularities, to which the alleged tetanus might have been due (?), and one man was said to have had a similar attack four years before. The remarks as to the treatment of erysipelas apply to that of tetanus as well.

Anthrax.—That anthrax should complicate ty-

[1] Simoneau, Jour. de Méd. de l'Ouest, 1882, 8.

[2] Fussell, Phila. Med. Times, Jan. 13, 1883, 263.

[3] Fowler, Buffalo Med. and Surg. Jour., 1880–81, xx, 155.

[4] Wolfinger, Ann. Städtl. Allgem. Krankenh., München, 1878, i, 44.

[5] Belhomme, Arch. de Méd. et Pharm. Milit., 1890, No. 6, 464.

phoid fever is quite unexpected. Only one case is recorded, that of a soldier[1] who died after having had intestinal hemorrhages and with enlarged spleen. In the stools during life, and in the blood, the intestines, the liver, and the spleen after death, were found the bacilli of anthrax. The stomach and intestines showed a number of tumors; Peyer's patches and the solitary follicles were eroded and the mesenteric glands enlarged; the spleen was five times the normal size. In the cecum and ileum were found the bacilli of typhoid.. The anthrax infection was traced to some milk his sister had brought him from a cow which had evidently suffered from anthrax.

Malignant Edema.—Brieger and Ehrlich[2] report two cases in which malignant edema complicated typhoid. They attribute it to the use of the tincture of musk for collapse.

The first was a woman of twenty-six. On the thirteenth day the musk was injected in the right thigh by a hypodermatic syringe. In two days there was severe pain and rapidly spreading gangrene, and she died the next day. The other was a woman of thirty-two in whom the same tincture was injected in the thigh with a similar result. In both cases the bacillus of malignant edema was found.

[1] Karlinski, Berlin. klin. Wochen., 1888, No. 43, 866.
[2] Berlin. klin. Wochen., 1882, 661.

CHAPTER XX.

OCULAR COMPLICATIONS OF TYPHOID FEVER.

BY GEORGE E. DE SCHWEINITZ, A.M., M.D.

History.—Disregarding for the moment the specific diagnosis,—typhoid fever,—a word in regard to post-febrile ocular complications in general may be in place.

In 1826 Dr. A. Jacob[1] described iritis as a sequence of relapsing fever, as this was first established by Hewson in his work on Venereal Ophthalmia. About the same time William Wallace[2] contended that this inflammation could be cured by "bark or quinin." In 1846 Dr. Jacob[3] returned to this subject in a communication entitled "On Inflammation of the Eye following Fever." His paper refers especially to a form of fever epidemic in Glasgow in 1843, and similar to that which prevailed in Dublin in 1826. This fever was described by Mackenzie,[4] and the ocular complications designated Post-febrile Ophthalmitis. They manifested themselves in a form of irido-choroiditis. Of 135 cases analyzed by A. Anderson, of Glasgow,[5] 10 began during the fever or its relapse; 34 began at once on convalescence; 29 within a fortnight of convales-

[1] Trans. Coll. of Phys. Ireland, Dublin, 1828, v, 468–478.

[2] Medico-Chir. Trans., London, 1828, xiv, 286–322.

[3] Dublin Med. Press, 1846, xv, 17–21.

[4] London Med. Gazette, n.s., vi, 1843–44, 225–236.

[5] Month. Jour. Med. Sc., London and Edinb., 1845, v, 729–773.

cence; 31 within the following month; and 31 within five or six months.

Similar severe inflammation of the eye following typhus fever, as it appeared in New York in 1847–48, has been reported by A. Dubois,[1] and again by Wilkes and Dubois.[2]

In 1870 post-febrile ophthalmitis received consideration from M. Charteris,[3] who investigated the cases of relapsing fever which occurred in Glasgow in 1870, and which, both from the ocular and general standpoint, were identical with those reported in the communications already referred to. The ophthalmic disease, as already stated, was an irido-choroiditis, which, if neglected, passed into ophthalmitis.

In 1878 Dr. N. Larionow[4] examined 767 typhus patients and typhus convalescents in order to ascertain the influence of the typhoid process upon the organs of vision. Among the ocular lesions which he described were conjunctivitis, iritis, keratitis, vitreous opacities, retinitis, neuritis, and "amblyopia and amaurosis."

From the preceding paragraphs it is evident that post-febrile ocular disease has been known for a long period of time as a complication of typhus and recurrent fevers, but less commonly of typhoid fever, although Larionow especially mentions typhus abdominalis.

Classification of the Ocular Complications and Sequelæ of Typhoid Fever.—In an article on the

[1] Trans. Amer. Med. Assoc., Phila., 1848, i, 373–386.

[2] Annalist, N. Y., 1848, ii, 331–333.

[3] Glasgow Med. Jour., 1870–71, 4th series, iii, 347–354.

[4] Klin. Monatsbl. f. Augenheilk., Cassel, 1878, xvi, 487–497.

alterations of vision in typhoid fever, Galezowski[1] submits the following classification: (1) Necrosis of the cornea; (2) thrombosis of the ophthalmic and orbital veins; (3) embolism of the central artery of the retina; (4) optic neuritis, with atrophy of the disc; (5) defects of accommodation.

An examination of the literature justifies a more elaborate classification, namely: (1) Affections of the conjunctiva and cornea; (2) affections of the uveal tract—iris, ciliary body, choroid—and of the vitreous humor; (3) affections of the crystalline lens; (4) affections of the optic nerve, retina, and retinal vessels; (5) affections of the orbit and orbital circulation; (6) affections of the extra- and intra-ocular muscles. Although not strictly ocular affections, to this list may be added affections of the cavernous sinus from extension of a diseased orbital process, affections of the sympathetic, and sloughing of the lids in association with noma of the face.

Relative Frequency of the Ocular Complications of Typhoid Fever.—If feebleness of accommodation as part of a general post-febrile weakness is excepted, affections of the conjunctiva and cornea are the most frequent ocular complications of typhoid fever. The cases of conjunctivitis (catarrhal and phlyctenular) far outnumber those of ulcerative keratitis. Suppurative keratitis, while fortunately not a common complication,[2] occurs with sufficient frequency to accord it more attention than it has thus far received.

[1] L'Union Méd., 1877, 3d series, xxiii, 937–941.

[2] August Hoelscher (Münch. med. Wochen., 1891, xxxviii, 43; 62) has seen it twice in 2000 fatal cases; Osler does not even mention its occurrence.

The remaining ocular lesions occur probably in the following order of frequency: Retinal hemorrhages, diseases of the uvea and vitreous, paralyses of the ocular muscles, neuritis and neuro-retinitis, and, finally, orbital affections. It is not unlikely that if ophthalmoscopic examinations were systematically undertaken in typhoid fever, the relative frequency of retinal hemorrhages would rise, and, indeed, the relative frequency of fundus lesions in general, because they attract attention only when they are so situated that they involve the macular region and occasion disturbances of visual acuity of which the patient complains.

I. Affections of the Conjunctiva and Cornea.—Ordinary conjunctivitis of the catarrhal type, differing in no sense from those varieties which are common to a number of febrile states, is frequent in typhoid fever. According to Knies,[1] during convalescence, and still more at a later period, phlyctenular affections, both of the cornea and of the conjunctiva, have been observed.

Of more serious import are the various types of ulcerative keratitis and sloughing of the cornea, occurring usually during the convalescent period, or, at all events, after the disease has existed for a sufficient length of time to have seriously depressed nutrition. Ulcer of the cornea may be preceded by the development of phlyctenules, or it may appear without such antecedents and rapidly pass into the sloughing type of keratitis with all its consequences.

[1] Die Beziehungen des Sehorgans und seiner Erkrankungen zu den übrigen Krankheiten des Körpers und seiner Organe, Wiesbaden, 1893, p. 395.

Cases of this character are reported by Knies,[1] Hoelscher,[2] Galezowski,[3] Adolph Alt,[4] and a number of other observers. The following case histories illustrating types of this affection are appended:

Case LVIII.—Hypopyon Keratitis with Iritis Occurring in the Fourth Week of Typhoid Fever.—Notes furnished by Dr. C. A. Veasey, of Philadelphia, as follows: A married woman in the fourth week of typhoid fever, complicated with pneumonia, developed a deep ulcer at the upper and inner quadrant of the cornea, together with iritis and hypopyon. Under atropin, boric acid lotions, irrigations with formaldehyd, and, finally, the application of the actual cautery to the ulcerated surface, healing took place with the preservation of vision of $\frac{5}{6}$, the cornea being clear except for a scar marking the position of the ulcer. There was one relapse of this ulcer two and one-half weeks after the original treatment had been begun, which rapidly yielded to the same measures.

Case LIX.—Ulceration of the Cornea Followed by Rapid Sloughing of this Structure, Occurring at the End of the Fourth Week of Violent Typhoid Fever.—Notes furnished by Dr. W. S. McClanahan, of Woodhull, Ill., as follows: A man, age twenty-three, was first seen on October 9th, with typhoid fever, his temperature at that time being 103.5°. The symptoms were severe from the very beginning, and in the third week the patient suffered from violent intestinal hemorrhages. Diarrhea, rapid emaciation, and delirium were prominent symptoms.

Ulceration of the right cornea was first noticed toward the end of the fourth week, beginning in a small point of infiltration near its lower margin. In

[1] *Loc. cit.* [2] *Loc. cit.* [3] *Loc. cit.*

[4] Med. Fortnightly, 3, 4, 1893, p. 47.

five days the entire cornea had passed into a state of ulceration, and the anterior chamber had filled with pus. Spontaneous rupture of the cornea and prolapse of the iris followed. Surgical interference was forbidden. When last seen by the reporter the inflammatory conditions were subsiding, although vision was practically *nil.*

The question of medico-legal responsibility in such destructive lesions of the eye has recently been brought to the attention of the courts, and, according to the Journal of the American Medical Association,[1] has been decided as follows:

"*No Duty to Provide Specialist.*—In the case of Jones *vs.* Vroom, decided by the Court of Appeals of Colorado, May 11, 1896, suit was brought against a firm of physicians to recover damages for the loss of an eye [from an ocular complication the result of her typhoid fever], alleged to have been caused by the negligent and unskilful treatment of the defendants. They had been employed to treat the plaintiff for typhoid fever. There was no evidence, and in fact no complaint, that they did not bestow upon her all the attention and skill which the nature of the disease and her condition required. Indeed, she stated herself that she was cured of the fever as a result of their treatment. The only charge in the complaint which was proven was that one of the defendants failed to send her an oculist after he had promised to do so. The Court holds that, under the circumstances, a nonsuit was properly granted. It says that the defendants were employed to treat the plaintiff for fever, and their employment imposed no duty upon them to provide her with a specialist for her eye. The Court took into account, furthermore, that she seemed to have

[1] July 18, 1896, p. 169.

had no difficulty in procuring one when she set about it, and says that presumably he could have been gotten just as readily at first."

According to Charles Stedman Bull,[1] who quotes Knies, in the somnolent or comatose stage of severe and fatal cases of typhoid fever, a rare complication is a true *xerophthalmia*, or *xerosis corneæ*. The same affection has been observed in cholera, diarrhea, and meningitis.

The *treatment* of the conjunctivitis of typhoid fever does not differ from that which is applicable to a similar conjunctivitis occurring under other circumstances—namely: a mild antiseptic lotion—for example, boric acid, or formalin, 1 : 6000. Severe corneal complications demand those measures which are suited to sloughing ulcers of other origins—namely: irrigation of the conjunctival cul-de-sac with a solution of formalin, 1 : 2000, or saturated boric acid, or bichlorid of mercury, 1 : 8000, atropin drops to control complicating iritis, and, if the ulcer extends, touching its surface, after the sloughing material has been curetted away, with a solution of nitrate of silver, ten grains to the ounce, or tincture of iodin, or, in severe cases, the actual cautery. If hypopyon develops, it should be evacuated by a paracentesis of the cornea. Powdered iodoform may be dusted upon the corneal surface with advantage.

The *prognosis* of suppurative keratitis is very bad, especially when it appears in aggravated cases of fever, or when the general treatment of the typhoid

[1] Med. Record, New York, 1897, vol. li, p. 577.

fever has been neglected; as, for example, in the cases reported by Adolph Alt, which occurred in very poor patients who had been unable to obtain proper attention. Under the best of circumstances so happy a result as that achieved by Dr. Veasey may be accomplished; under other circumstances, even with the most careful treatment, extensive sloughing of the cornea may result as in the case recorded by Dr. McClanahan.

II. Affections of the Uveal Tract—Iris, Ciliary Body, Choroid—and the Vitreous Humor.—As may have been inferred from the earlier portion of this chapter, affections of the uveal tract are much more frequent in relapsing and typhus fever than in pure typhoid fever. None the less, iritis, cyclitis, and choroiditis, either in the serous or the plastic form, and associated with opacities of the vitreous, are occasionally encountered during typhoid fever, especially during convalescence. F. Sorel,[1] in an analysis of 871 cases of typhoid fever observed during a period of ten years, notes only one case of iritis during the period of convalescence. In spite of the apparent infrequency of inflammation of the iris, or of the uveal tract generally, I am persuaded that more minute examination of the visual organs during typhoid fever would reveal a greater prevalence of affections of this tract than might be inferred from such statistical information as has just been quoted. Alt has seen plastic choroiditis with opacity of the vitreous after a severe attack of typhoid fever, and I have observed post-typhoidal choroiditis and secondary atrophy of the disc.

[1] Bull. et Mém. de la Soc. Méd. des Hôp., 1889, vi, 3, s., 224–246.

The floating opacities which occur in the vitreous, according to Bull, are of the punctate or stringy variety, but are not fixed and membranous.

There is some evidence to show that **spontaneous inflammation of the vitreous,** which may manifest itself simply as an opacity, or even go on to suppuration, may be due to the exhaustion and debility consequent upon a low fever like typhoid, and, as Howard F. Hansell has pointed out, if during the earlier stages of the fever the discovery is made that fine flakes of opacity are beginning to appear in the vitreous, it is possible that a vigorous supporting treatment may save the eye from destruction. Although Bull states that these uveal tract inflammations are serous or plastic in type, but never purulent, it would seem that this rule is subject to exceptions, and that a purulent choroiditis similar to that which is seen in pyemia, puerperal sepsis, or endocarditis, may occasionally complicate typhoid fever. A case in point is the following:

Case LX.—Panophthalmitis Occurring at the Beginning of the Third Week of Typhoid Fever and Resulting in Phthisis Bulbi. Notes furnished by Dr. C. W. Hall, of Kewanee, Ill. A woman, age thirty, was taken ill with typhoid fever on October 29, 1895. During the first week headache was severe, the temperature ranged between 101° and 104° F., and there were marked tympanites, abdominal tenderness, and moderate diarrhea. On the sixteenth day throbbing pain began in the left eye, quickly followed by loss of vision. In a few days the symptoms of panophthalmitis were marked, especially edema of the lids and chemosis of the conjunctiva, and, in fact, swelling of all the tissues surrounding the eye. The

eye gradually grew worse and sloughing of the ocular tissues supervened, followed by atrophy of the eyeball, which gradually shrank to the size of a large hazelnut.

In this case, as there is no report of external lesion of the cornea, the disease beginning with pain and loss of sight, we may assume a purulent choroiditis caused by embolism from a microbic area.

The *treatment* of cases of uveal tract inflammation must be governed by the type of the disease. In iritis the mydriatics are indicated, and the same remedies would be advisable in inflammations of the deeper structures. Success would depend, as has already been pointed out, upon being able to support the nutrition of the patient. If purulent choroiditis should arise, followed by panophthalmitis, incision of the eyeball and evisceration of the purulent contents would be required.

III. Affections of the Crystalline Lens.—As the result of inflammation of the uveal tract, the nutrition of the lens is interfered with and cataract may result. Without such antecedent inflammation, opacity of the crystalline lens has been attributed to the nutritional disturbances occurring during typhoid fever. For example, Trélat[1] has described double, semi-soft cataracts in a young girl, which began to develop during convalescence from typhoid fever, and Fontan[2] reports three cases of post-typhoid cataract (cataracta punctata) which he believes were the result of mechanical obstruction of the circulation. In two of these cases, one a twenty-eight-year-

[1] Gaz. des. Hôp., 1879, p. 417.

[2] Rec. d'Ophtal., 1887, 3, s., ix, 195-200.

old man, and the other a forty-two-year-old woman, the cataract progressed to maturity and required extraction. Unfortunately, there is no evidence to show that Fontan was aware of the condition of the transparent media before the typhoid attack. Romiée[1] has analyzed 44 cases of cataracta punctata, and attributes the pathogenic cause to typhoid fever in 17 of them. He seeks to explain the lenticular opacity by an increase in the density of the serum and changes in the relation of the lens to the aqueous humor. It is most likely that all cases of post-febrile cataract are originally due to disturbances of moderate or greater grade in the uveal tract, necessarily followed by changes in the nutrition of the lens. An interesting surgical fact in connection with these cases is that they seem to do as well after extraction, as those cataracts which have not developed through such a complication.

IV. Affections of the Optic Nerve, Retina, and Retinal Blood-vessels.—Double **optic neuritis** may occur and end in atrophy of the nerve or in recovery; as, for example, in cases recorded by Nothnagel, Hutchinson, Sr., Clifford Allbutt, and in our own country by Augustus P. Clarke[2] and J. A. White.[3] The last observer not only gives his own experience in this particular, but has gathered the observations of a number of American ophthalmic surgeons who have from time to time noted cases of neuritis and optic nerve atrophy attributed to typhoid fever.

[1] Rec. d'Ophtal., 1879, 3, s., i, 586–593.

[2] Jour. Amer. Med. Assoc., 1891, xvi, 473–476.

[3] Trans. Ophth. Section Amer. Med. Assoc., Chicago, 1893, 215–223.

Some doubt in regard to the reality of this typhoid neuritis has been expressed, especially by Leber and Stellwag von Carion, who have suggested that the cases in which it occurred may have been meningitis instead of typhoid fever. Commenting on this, however, Gowers points out that neuritis does occasionally follow other acute specific diseases. Neuritis of other nerves than the optic has been observed in typhoid.

Robert P. Oglesby [1] has never met with a case of optic nerve trouble after typhoid fever unless there have been symptoms of meningitis. In his opinion, the condition is not one of actual neuritis, but of so-called subacute neuritis. According to this author, cases of typhoid fever amblyopia are more frequent among women than among men, and especially among child-bearing women. A history of cerebral neuralgia and meningitis can always be obtained. Gowers, on the other hand, referring to the fact that cases of typhoid fever accompanied by hyperemia of the discs have been supposed to be cases complicated by meningitis, points out that meningitis, except as secondary to suppuration in the ear, is exceedingly rare in this disease, and that it is not warranted to infer meningitis because there is extreme delirium or coma. Meningitis not arising from ear disease, but from direct microbic infection, however, is recorded by Keen in Chapter VIII.

Braine-Hartnell [2] has reported a case which proves that bilateral optic neuritis may complicate typhoid

[1] Brain, London, 1882, v, 197–203.

[2] British Medical Journal, May 29, 1897; also Medical Record, New York, vol. lii, p. 349.

fever when there is no meningitis. His patient was a boy, eleven years old, who suffered from fever, diarrhea, photophobia, cerebral irritation, retraction of the abdomen, dry tongue, and sordes on the teeth. There were no spots, however, and the spleen was not enlarged. There was never any strabismus, nor were convulsions present. Two days before death there was slight inequality of the pupils, and well-marked bilateral optic neuritis developed. Neither aural nor nasal discharge was detected. The post-mortem examination revealed distinct inflammation of Peyer's patches, with decided enlargement of the mesenteric glands and solitary follicles. Nothing was found in the brain to explain the optic neuritis.

In my examinations of typhoid fever cases in the Philadelphia Hospital, while I have seen hyperemia of the discs, I have never observed actual neuritis in cases uncomplicated with meningitis; in fact, in one case in which the diagnosis was somewhat uncertain, and which came to autopsy, the lesions of a disseminated tubercular meningitis were present, but no signs of the typhoid process. I have therefore come to regard optic neuritis as in a certain sense a valuable differential diagnostic point in cases of low fever of uncertain origin.

Optic nerve atrophy, either partial or complete, may follow the neuritis, or, either single or double, may be present without preceding inflammation. These cases are usually noticed first in the convalescent period and come under more exact examination as post-typhoidal phenomena. A matter of some importance in determining the etiology of cases of

post-febrile atrophy is to ascertain what treatment the patient has undergone, because it is well recognized that the excessive use of quinin during the fever may occasion amblyopia and even partial optic nerve atrophy. Intestinal hemorrhage may also be the exciting cause of permanent blindness from atrophy of the optic nerve, precisely as this occurs after hematemesis. Amblyopia has also followed severe epistaxis (Ebert, quoted by Knies) and menstrual hemorrhage during typhoid fever, as reported by C. Williams.[1]

Instead of intraocular optic neuritis, temporary or permanent impairment of vision may be due to **retrobulbar neuritis**, which may perfectly recover or eventuate in optic nerve atrophy. These cases have been referred to by Knies, and, as has been suggested by Bull, are difficult to study because the patient is usually too ill to permit perimetric examination, which, no doubt, would reveal a central scotoma. Occasionally the blindness may be due to a retrobulbar neuritis, caused, probably, by a hemorrhage into the optic nerve, as in a case reported by Leber and Deutschmann, which is quoted by Knies.

Amblyopia without ophthalmoscopic changes, similar to the blindness occurring after or during scarlet fever, has been recorded, for example, by Ebert[2] and by Nothnagel,[3] and presents a favorable prognosis, the affection, according to Gowers, usually passing away in the course of several weeks.

Leber has described **anesthesia of the retina**, and

[1] Archives of Ophthalmology, New York, 1884, xiii, 397-399.

[2] Berl. klin. Wochen., 1868.

[3] Deutsch. Archiv f. klin. Med., 1872, ix, 470.

Hersing an annular defect in the visual field. J. Stewart[1] has observed, in a case of typhoid fever in a man aged thirty-two, setting in with meningitic-like symptoms, an island-shaped scotoma. The symptoms were attributed to blood intoxication.

Neuro-retinitis with macular hemorrhages has been described by von Petershausen,[2] and Munier[3] has reported double blindness from neuro-retinitis. In his case there was probably a complicating meningo-encephalitis.

According to Charles Stedman Bull, **retinal hemorrhages** are by no means uncommon in the height of typhoid fever. They vary in size, shape, and appearance, according to the portion of the retina in which the extravasation takes place, and are said usually to appear about the third week of the disease. They may burst through the limiting membrane and involve the vitreous. Sometimes they manifest themselves in association with intestinal hemorrhage.

The exact cause of retinal hemorrhage in this disease has not been determined, but it probably depends, as Bull has suggested, either upon a weakened condition of the blood-vessel walls, or a perverted quality of the blood, or both combined. Sometimes, no doubt, there is rupture of the vessel wall itself and sometimes a diapedesis. A microbic invasion of the vascular walls has been suggested by Dr. Keen (pp. 60 and 172) as a possible explanation of these and kindred hemorrhages. I am con-

[1] Montreal Med. Jour., 1894-95, xxiii, 752-758.

[2] Detroit Rev. Med. and Phar., 1873, viii, 533-541.

[3] Thèse de Paris, 1874.

fident that retinal hemorrhages are much more common in typhoid fever than is generally supposed. The subject appears, in large measure, to have escaped the attention of clinicians.

Finally, a few cases of **embolism of the central artery of the retina** have been observed during the convalescence from typhoid fever; for example, by Galezowski[1] and Snell.[2]

V. Affections of the Orbit and Orbital Circulation.—Galezowski refers to the fact that Burgeois and Trousseau, among other complications, have reported **thrombosis of the orbital veins,** and Caron du Villards, **phlegmon of the orbit.** Orbital cellulitis as a complication of typhus is well known. It is possible that some of the cases may have been due to typhoid fever. Indeed, Swanzy and one or two other authors mention typhoid fever as a cause of inflammation of the orbital tissue, but give no details.

Panas[3] details the following remarkable case: A patient, seven years of age, who five years previously had been under treatment for an angioma of the orbit, developed, during the course of typhoid fever, in the third week, phlegmonous inflammation of the orbit and panophthalmitis. When the bulbus was enucleated an angioma was found, the center of which had undergone suppuration. In the midst of the suppurating tissue bacteriological examination developed the presence of the bacillus of Eberth. Panas points out that this process must be explained on the theory of endo-infection, the thrombotic con-

[1] *Loc. cit.* [2] Ophthalmic Review, i, 403.
[3] Congrès Français de Chirurgie, 5th session, Paris, 1891, 63–69.

ditions in the orbit presenting a particularly favorable field for the development of suppuration under the influence of the micro-organisms.

A most interesting case of **spontaneous orbital and intraocular hemorrhage** occurring in the course of typhoid fever has been reported by C. A. Finlay.[1] It occurred in a boy, ten years of age, in the third week of typhoid fever, and was associated with the appearance of ecchymotic spots, not only in the conjunctiva, but all over the body and limbs. The corneæ of both eyes sloughed. The patient recovered. Dr. Finlay has collected 26 cases of spontaneous orbital hemorrhage, but this is the only one which occurred during the course of typhoid fever. Finlay suggests that in his case there may have been some degenerative change in the walls of the blood-vessels in connection with the general lowering of the nutrition, or else the condition should be considered as a complicating purpura.

VI. Affections of the Intra- and Extra-ocular Muscles.—As has already been stated, during the period of convalescence dilatation of the pupil and **paresis of accommodation** is not an uncommon affection. Segal[2] describes mydriasis in typhoid fever with normal accommodation and normal vision, and considers the dilatation due to an irritation of the sympathetic.

Paralyses of the extra-ocular muscles during the height of typhoid fever, unless there is some grave intracranial complication, are exceedingly rare. Nothnagel has reported double ptosis and right

[1] Archives of Ophthal., New York, 1897, xxvi, 42–47.
[2] Archiv f. Augenheilk., Wiesbaden, 1889, xix, 386.

abducens paralysis at the beginning of the third week.

In the period of convalescence they have been observed more frequently, and, according to Knies, may perhaps be attributed to a chronic nephritis, which in his belief is a frequent sequel of typhoid fever. According to this author, these paralyses recover quickly and also relapse quickly, and are nuclear in character.

Finally, there may be muscular paralyses occurring at long periods after typhoid fever; for example, a case of left trochlear paralysis reported by Runeberg, which appeared one and one-half years after fever. The etiological relationship of the typhoid state to these palsies is very questionable. If there should be a complicating meningitis at the base of the brain, then, naturally, orbital muscle palsy might be expected, exactly as we find it in ordinary meningitis; and, indeed, cases of this kind are not infrequent. For example, Samuel West[1] observed during a mild attack of typhoid fever in a child, aged ten, retraction of the head and strabismus, which disappeared within a few days, the case resulting in recovery.

A complicating meningitis of the cortex, or at the base of the brain, might produce **hemianopsia**, which, according to Knies, is a very rare phenomenon.

[1] St. Barth. Hosp. Rep., 1886, vol. xxii, p. 224.

CHAPTER XXI.

CONCLUSIONS.

If now, by way of review, we cast our eyes back over the general results of all the complications and sequels we have studied, we may arrive at some useful and important conclusions.

1. *Typhoid*, probably from its usually longer duration, is by far the more prolific source of such surgical troubles, except parotitis, especially when we consider that many cases tabulated as typhus are really typhoid. Of 1699 cases, typhoid was the preceding fever in 1139, typhus in 494, and other forms of continued fever in 66. Of the 494 after typhus fever, 352 were cases of parotitis. In the paragraphs which follow I have entirely omitted these, as they would so warp the figures as to give false results. The statistics are therefore based nearly entirely upon the typhoid cases. The former tables not having been preserved, the typhoid and typhus cases in each lesion could not be discriminated; but besides the cases of parotitis there were only 119 in all.

2. The *surgical troubles* to be apprehended in *typhus*, apart from parotitis (352 cases), are mainly restricted to gangrene and laryngeal stenosis, 103 out of the 119 cases after typhus being due to these two classes of disease, while typhoid bears in its train any and all of the forms of disease described.

3. When the *infection* is a *mixed infection* of the typhoid bacillus and other bacteria, the result is a complication rather than a sequel, and the course of the disease is apt to be early in its onset, acute, and of a febrile character. When the infection is by the typhoid bacillus alone, the result is a sequel rather than a complication, and the course of the disease is apt to be late in its onset, chronic, and afebrile. In the pure typhoid infections the disease is often local rather than general, and the patient's general health suffers but little.

4. The *cause* of nearly all these surgical results (using this word to include both the complications and the sequels) of typhoid fever is the typhoid bacillus, and not, as I thought twenty years ago, the presence of a clot or of mechanical conditions. True, these mechanical conditions do influence the site of the lesions in many cases, as also does the existence of a clot, but the etiological factor of most importance is the bacillus itself. The bacilli have not so far been found generally in the joints, in parotitis, otitis media, or the laryngeal lesions, and often not in gangrene, abscesses, and hematomata; but their almost universal presence in the lesions of the bones, meningitis, abscesses, the gall bladder, and the spleen, would lead us to suspect their influence in the other cases, at least at some period of the disease. To many of the regions and organs of the body in which they are not usually found the pyogenic bacteria find a ready access, and undoubtedly exert a potent influence. But most of the surgical results can be attributed directly to the bacilli of the fever itself,

aided by other bacteria and by unfavorable mechanical conditions.

5. The *clinical course* of the surgical results of typhoid is worthy of a word. It is often peculiar in that the same complication or sequel affects multiple regions of the body—*e. g.*, a number of different bones—either simultaneously or successively; or, again, that two or more complications may attack the same patient—*e. g.*, gangrene and an osseous lesion, or a laryngeal complication, and abscesses in different regions of the body.

6. The *period of development* is not the initial period of the fever, but, first, from its height to its close—that is, the complications, especially gangrene and stenosis of the larynx; and, secondly and most frequently, during convalescence—that is, the sequels. Of 622 cases, only 12 arose in the first week, 113 in the second, and 78 in the third, a total of 203. If we may assume that convalescence, on the average, begins at the end of the third week, then 419 occurred during or after convalescence. Many of them occur months, and even years, after the fever. This is especially true of the lesions in the bones. That the typhoid bacilli may cause such late manifestations of disease has naturally been doubted. But the array of proof, bacteriological as well as clinical, in the preceding chapters would seem to be complete and to set at rest any lingering doubt.

The pure typhoid infections are, as a rule, later in their onset; the mixed infections occur much earlier.

7. The *lower half of the body* is the especial seat of such surgical troubles. With the exception, of

course, of the cerebral, laryngeal, aural, thyroid, and thoracic cases and parotitis, and the cases of biliary and intestinal perforations, of 942 cases of general disorders, such as gangrene, osseous lesions, etc., 515 occurred in the pelvic region and legs, as against 427 in all other parts of the body. Moreover, the diseases attacking the upper half of the body are limited almost entirely to local gangrenes and osteomyelitis, caries, and necrosis, and they are usually far less severe in type and more limited in extent than those in the lower half. Here whole limbs are blighted by gangrene; here occur most of the dislocations, the hematomata, the fistulæ; here the severest necroses and largest abscesses; and were we to add the long catalogue of bedsores and phlegmasiæ, the preponderance of the lower half of the body in importance would be still further increased.

8. The *age* is about the usual age of greatest frequence of these fevers.[1] From fifteen to twenty-five years is by far the most frequent decade, count-

[1] Liebermeister gives the ages in typhoid as follows:

Fifteen to thirty,	1,310
Thirty to seventy-one,	394
Total,	1,704

None under fifteen were admitted.

In typhus:

Fifteen to thirty,	39 per cent.

Murchison gives in typhoid:

Fifteen to twenty-five,	2,752
All other ages,	3,159
Total,	5,911

And in typhus:

Fifteen to twenty-five,	5,332
All other ages,	12,806
Total,	18,138

ing 361 cases against 353 at all other periods of life. One singular exception is to be made—viz., the articular troubles, and especially dislocation of the hip, 32 out of 35 cases being under twenty years of age, of which 21 were in children under fifteen—in striking analogy to the frequency of coxalgia in children.

9. *Sex* is an unexpected and important factor in the predisposition to febrile surgical troubles. Of 745 cases in which the sex is named, 488 are males and 257 are females, or nearly two to one. What is the normal proportion of the sexes in fever it is difficult to determine. In nearly 6000 cases of typhoid, Murchison gives the proportions as precisely equal, and in over 18,000 cases of typhus the females were in a decided majority (8871 to 9267). Estlander's figures would give us a slight preponderance of males, while Liebermeister, in over 2000 cases of typhoid, gives 1300 males and 750 females.

Unfortunately, I omitted to tabulate the number of cases arising in *military practice*, which I am sure is not inconsiderable; but while this will account to some extent for the predominance of males, it could not be adduced in the cases of arthritis and dislocation, since most of these patients were children, yet the males were in the preponderance.

10. To the surgeon the most interesting recent developments are those relating to *biliary and intestinal perforation*. We may even take courage as to these formerly almost uniformly fatal complications. Of four cases of perforation of the gall-bladder thus far submitted to operation, three have recovered. Nat-

urally, the outlook in intestinal perforation is far less encouraging, but it is far from hopeless. Of 83 cases operated upon, 16 have recovered, or 19.3 per cent. If, knowing the possibility of recovery, physicians more frequently call in the surgeon, and do so at once, the future will show better results.

I can not too strongly urge that the essential mortality of operations for typhoid intestinal perforation is not accurately estimated by the mortality of *all* the cases (80.7 per cent.). *The real mortality thus far is only 69.4 per cent.; that is to say, the mortality of the cases operated on within practically the first twenty-four hours.* After that nearly all hope is gone. The table on page 227 shows that, including case 64, operated on after twenty-six hours, there were, within this period, 36 cases with 11 recoveries (30.6 per cent.). *When every case of intestinal perforation (except the moribund) is operated upon within twenty-four hours, I believe that we shall save at least one-third of the cases and possibly more.*

11. The *diagnosis* is, in general, moderately easy. The danger is not that difficulty of diagnosis may obscure the case, but that the diseases may be entirely overlooked. They occur most frequently in parts of the body covered by the bedclothes; parts which require time and trouble to expose and examine in the routine of an ordinary visit. Moreover, the patient is frequently so apathetic and insensible to pain that he does not complain, or, if he do so, it is ascribed to the ordinary pains so frequent in the belly and legs in such fevers, or else to the delirium itself.

Hence, the most important hint I can give in the

diagnosis—and where, indeed, does the same rule not hold good?—is that time and trouble *must* be taken, and that no patient suffering from a continued fever, and especially from typhoid, should escape frequent, minute, complete, physical examinations, in which every part of the body from head to foot should be questioned. Especially should the physical condition of the larynx, the belly, the legs, and the toes, and, in children, the hip-joint, be exactly ascertained. This should be done at least every second day; and that, too, not only in severe, but in mild cases, and not only during the fever, but especially in early convalescence, for it is in just such mild and convalescent cases that the wariness of the doctor is the patient's surest reliance. Particularly should attention be paid to hoarseness or even the slightest change in the voice, and if these be present the larynx should be examined at once with the greatest care, from day to day, by the eye, the finger, and the laryngoscope, lest sudden edema or the more insidious and more fatal necrosis of the cartilages be impending. The eye should seize upon any hindered movements, even without discomfort, and no complaint of pain should fall upon a deaf ear, especially if it be in the throat, the belly wall, the buttock, the hip-joint, the legs, or the toes. True, it may mean nothing. It may be the vagary of a wandering mind. But it may also be, as we have seen, the herald of the gravest dangers, whose attack may be entirely repelled or their force broken by heeding this timely warning.

12. The *prognosis* is naturally unfavorable, yet not to the extent we would suppose from the addition or

sequence of such serious disease. Of 1037 cases in which the result is named, 456 died and 581 recovered—a mortality of 44 per cent.

13. The *treatment* must be bold, but not rash; conservative, but not timid; prompt, but not hasty; thorough, but not reckless.

POSTSCRIPT.

The following cases have been published since the foregoing pages were already in print, and are added to make the treatment of the subject as complete as possible.

Dr. Hodenpyl's paper, referred to on page 45, has been published in the British Medical Journal for December 25, 1897, page 1850.

Typhoid Infection without the Intestinal Lesions of Typhoid Fever (page 43).—Chiari,[1] before the Pathological Section of the Moscow International Medical Congress, read a paper on cases of typhoid infection without infection of the mesenteric glands or the intestine. He characterizes such cases as "typhus-septicemia." He reported the following case:

Case LXI.—The Gruber-Widal serum reaction confirmed the diagnosis of typhoid, but at the necropsy no characteristic intestinal changes were found, but the bacteriological investigation showed the presence of typhoid bacilli.

[1] Centralbl. f. alleg. Pathol. and patholog. Anat., Oct. 15, 1897, viii, 790.

Case LXII.[1]— A man, age twenty-five, was admitted to the hospital, July 8, 1897, as an ambulant case of typhoid fever. He had suffered with headache, loss of appetite, and general weakness. The abdomen was distended, tense, and tender. Fading rose spots were visible. The spleen was palpable; bowels constipated. The pulse was dicrotic. The blood gave the typical serum reaction. From the 8th to the 13th he was semi-comatose. Just before death his temperature reached 106.6°. Cultures made from the blood the day before death were sterile. He died on the 21st. The necropsy was made eight hours later by Drs. Adami and Nicholls. The mesenteric glands, especially those about the ileo-cecal region, were enlarged, congested, and succulent. "The lowest three Peyer's patches of the ileum were very slightly raised above the general surface, but showed no signs of inflammation. The remaining Peyer's glands were normal. There was no evidence of ulceration anywhere in the intestine nor any evidence of healed typhoidal lesions." By the microscope, clumps of the typhoid bacilli were found in the intestine and in the mesenteric glands. A section through one of Peyer's glands which presented slight swelling showed small clumps of bacilli resembling typhoid. The blood-serum taken at the necropsy gave the typical serum reaction. Cultures from the spleen and the liver showed the typhoid bacillus and reacted to the serum-test. All these tests were made by Nicholls and Adami independently, and agreed exactly.

Osteomyelitis of the Skull and Abscess of the Brain (Chapter VIII, p. 169).—The following

[1] Nicholls and Keenan, Montreal Med. Jour., Jan., 1898, p. 9.

case of presumed osteomyelitis and abscess of the brain following typhoid fever occurred in 1889, but, owing to its defective title, I discovered it too late to insert it in the proper place.

Case LXIII.—Terrillon[1] reports the following case: On December 24, 1888, Richard was called to see a boy of thirteen with violent pain in the head, vomiting, and intense fever. After eight days his sickness apparently ceased, and it was believed that he had been ill of scarlet fever. Five days after defervescence—*i. e.*, January 3, 1889—he was seized with convulsions. The urine showed no albumin. The fever and the pain in the head returned, and he was delirious for five or six days. Richard was then convinced that the child's illness was typhoid fever. On the 20th of January convalescence began, and appeared to be complete by February 1st. On February 14th he began to complain of pain in the left temple, where a slight swelling was found. On the 20th an incision was made, which evacuated only blood. On the 21st, aphasia and paralysis of the right arm were observed, with a temperature of 38° C. Terrillon first saw the child February 23d. He found marked tumefaction in the left temple, with fluctuation and surrounding edema. The boy was in a profound torpor. Paralysis of the right face and arm was still evident. An incision was immediately made over the left temple, evacuating a considerable amount of thick, grumous pus. The bone was denuded, and the base of the cavity lined with fungous granulations. On the 25th a temporary improvement which had taken place disappeared. The child was immediately trephined. The dura was not opened, but three punctures were made, and at the third, passing forward and downward, more than a tablespoonful

[1] Bull. et Mém. Soc. de Chir. Paris, 1889, n. s. xv, 555.

of greenish, creamy pus was evacuated. An incision was then made and drainage established. On awakening from the anesthesia he could move the right arm, and the facial paralysis had almost entirely disappeared. He was able also to pronounce several words. In spite of this apparent improvement, however, he soon became worse, and died on the 3d of March, five days after the operation. The conclusion of Terrillon was that an osteomyelitis of the bones of the skull had given rise to both of the abscesses.

Two very unfortunate omissions, however, are notable in the case: First, no necropsy was made, so that while the case was presumably one of typhoid, it is by no means certain; and, secondly, and still more important, no bacteriological examination was made, although the bacillus of Eberth had been demonstrated as the cause of typhoid nine years before.

Suppuration Around the Pituitary Body with Thrombosis of the Right Sylvian Vein and Both Cavernous Sinuses (Chapter VIII, p. 169).

Case LXIV.—The following case of this very unusual condition after typhoid fever is kindly furnished me by Dr. Joseph Sailer, of Philadelphia. A young man, twenty years of age, suffered a moderate attack of typhoid fever in the autumn of 1897, when under the care of Drs. Henry Morris and G. M. Marshall in St. Joseph's Hospital, Philadelphia. All the characteristic symptoms had been present, including the Widal serum reaction, at three different examinations. During defervescence he was suddenly attacked by a severe chill, which lasted with intermissions for about forty-eight hours. During this period and three days later the Widal reaction was negative. Subsequently it reappeared, and was readily obtained in a dilution of 1 to 30. A few

days after his chill, swelling was noticed in the right parotid gland. This rapidly increased, and in a short time the patient developed bilateral exophthalmos, with considerable subconjunctival effusion. Fluctuation was then detected in the parotid gland; the gland was incised and several ounces of extremely fetid pus removed. This was given me [Dr. Sailer] for examination, and found to contain a great variety of micro-organisms. It was not possible to isolate any, however, that resembled the typhoid bacillus. Death occurred two days later. I made the autopsy within five hours; found a few typhoid ulcers in the ileum which were beginning to heal; a purulent exudate in the left pleural cavity, also due to a mixed infection, and a suppurative condition about the hypophysis cerebri, with thrombosis of the right Sylvian vein and both cavernous sinuses. Both retro-bulbar spaces contained a large effusion of liquid. Cultures of the bacillus pyocyaneus were obtained from the mesenteric glands. In the thrombi of the cerebral veins a number of species of micro-organisms were found, all of which have not been identified, but none of which resembled the typhoid bacillus. Cultures from the spleen were negative.

Cholecystitis following Typhoid Fever (Chapter XVI, p. 244).—The following case of probable perforation of the gall-bladder has also been published by Dr. James Bell in the Montreal Medical Journal for January, 1896.

Case LXV.—Miss C., age thirty-five, at the end of the second week of typhoid fever was seized with pain, vomiting, and a fall of temperature to 94.5°, early in the morning on September 21, 1897. The abdomen was opened September 24th, at 4.30 P.M. There was no general peritonitis, but the

gall-bladder was distended and covered with patches of lymph, as also were the lower border of the liver and adjacent coils of intestine. There was no perforation. By aspiration he withdrew six ounces of pus, which showed the typhoid bacillus in pure culture. The gall-bladder was then incised and 152 gall-stones removed; a drainage-tube was inserted and bile flowed freely. In the lower angle of the abdominal wound a drainage-tube and iodoform gauze packing were carried up along the under surface of the liver. She did well until the morning of the 26th, when symptoms of perforative peritonitis set in, and she died at 4 A.M. on the morning of the 27th. An incomplete post-mortem examination showed a general purulent peritonitis, and four gall-stones were removed from the lower angle of the wound. The small intestine showed the typical lesions of typhoid. "In the absence of a complete post-mortem examination of the abdomen, my [Dr. Bell's] interpretation of the sudden termination in this case is that ulceration of the deeper part of the gall-bladder or of the cystic duct had taken place into or upon the adjacent under surface of the liver, and that it was the bursting or emptying of this abscess cavity which set up the fatal peritonitis. In no other way can I account for the presence of the four gall-stones in the peritoneal cavity, as I am sure they did not escape into the abdomen at the time of the operation, and the steady flow of bile externally for hours after the fatal symptoms set in showed that there could not be any direct communication with the abdominal cavity. Moreover, the wound was examined and the gall-bladder sutures found intact five hours after the onset of the symptoms."

Leukemic Spleen following Typhoid Fever (Chapter XVII, p. 274).—While correcting the

proofs, the following case, almost the duplicate of Case XLIX, page 275, came under my own observation. Figure 4, illustrating that case, was practically exactly duplicated in the present case.

Case LXVI.—E. L. P., of Mt. Holly, N. J., first consulted me, with Dr. R. H. Parsons of the same place, January 22, 1898. His age is thirty-three; his average and present weight is 124 pounds. Three years ago he only weighed 95 pounds. His father is living and in good health; his mother died of hemorrhage after a miscarriage. His paternal grandmother died of hemorrhage of the lungs at seventy-three; his paternal grandfather is still living at ninety. His maternal grandmother died at eighty-one of old age; his maternal grandfather of some unknown disease. For some two or three years he has suffered with alternate constipation, when he would often not have a movement for a week, and a loose condition of the bowels, when he would have two or three movements a day; the latter gave him much relief. The patient has suffered from several attacks of grippe, notably a very severe attack in the spring of 1895. This was followed by typhoid fever, and from July to October, 1895, by an attack of inflammatory rheumatism. On his recovery from the inflammatory rheumatism his weight was only 95 pounds. In July, 1895, while under treatment for the rheumatism, and shortly after his attack of typhoid fever, his attending physician discovered a lump in his left side, and pronounced it an enlarged spleen. Not long after his recovery from the rheumatism he had severe pain in the abdominal tumor, "so severe that he was almost crazy" for two or three weeks. From January to March, 1896, he was under the care of Dr. William Pepper, of Philadelphia, who confirmed the diagnosis of an enlarged and leukemic spleen. He has never

had any blood in the urine or other indication of trouble with the kidney. For some time past he has been suffering daily from fever, his temperature rising to 101° or 102°. In spite of this, however, ever since passing from Dr. Pepper's care he has been attending to his business, and, on the whole, seems not to have lost much ground. An examination of the abdomen shows a tumor almost identical with that shown in figure 4, page 276. An examination of the blood made by Dr. Kyle gives the following result:

Hemoglobin, ...	70 per cent.
Red corpuscles per cubic mm., . . .	4,066,000
" " " " normal,	3,000,000
White corpuscles per cubic mm., . . .	200,000

The case being one of leukemic spleen, I declined to interfere surgically.

APPENDIX.

TONER LECTURE, NO. V.

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INSTITUTED TO ENCOURAGE THE DISCOVERY OF NEW TRUTHS
FOR THE ADVANCEMENT OF MEDICINE.

LECTURE V.

ON THE SURGICAL COMPLICATIONS AND SEQUELS OF THE CONTINUED FEVERS.

BY

WILLIAM W. KEEN, M.D.,
OF PHILADELPHIA.

DELIVERED FEBRUARY 17, 1876.

WASHINGTON:
SMITHSONIAN INSTITUTION.
MARCH, 1877.

ADVERTISEMENT.

THE "Toner Lectures" have been instituted at Washington, D. C., by Joseph M. Toner, M.D., who has placed in charge of a Board of Trustees, consisting of the Secretary of the Smithsonian Institution, the Surgeon-General of the United States Army, the Surgeon-General of the United States Navy, and the President of the Medical Society of the District of Columbia, a fund "the interest of which is to be applied for at least two annual memoirs or essays relative to some branch of medical science, and containing some new truth fully established by experiment or observation."

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JOSEPH HENRY,
Secretary Smithsonian Institution.

SMITHSONIAN INSTITUTION,
Washington, April, 1877.

LECTURE V.

Delivered February 17, 1876.

ON THE SURGICAL COMPLICATIONS AND SEQUELS OF THE CONTINUED FEVERS.

By William W. Keen, M.D.,
of Philadelphia.

The province of the physician and that of the surgeon are, in general, sufficiently sharply defined and differentiated, yet they have many points of contact. While some diseases belong exclusively to the province of the one, and some to that of the other, other diseases may fall with equal propriety under the care of either practitioner. Still another class of cases, however, beginning in the domain of medicine, may terminate in that of surgery, and we may lack their complete history from the very fact of this division of their care and interest.

Among the diseases classed as strictly medical, none deserve the appellation more definitely than the continued fevers, and especially typhus and typhoid. Yet I hope to show that fevers are not infrequently the cause of the gravest and least expected surgical troubles, mention of which is generally omitted, even in our best text-books on medicine, still more rarely noticed in works on surgery, and, where noticed, it is only with the greatest brevity.[1]

[1] "The cases of constitutional disease discovered by fever might serve to illustrate a large part of the convalescence of fever, a subject of the highest interest and full of promise of utility to one who will carefully study it. The sequelæ of scarlet fever are commonly enumerated; those of typhoid fever, especially those seen in surgical practice, are scarcely less numerous, but seem less known." Just as this is going to press, I find the above remarks by Sir James Paget, in his extremely interesting Clinical Lectures and Essays, London, 1875, p. 378.

My attention having been called to the matter by several personal cases, I have been led to study the subject, and I desire now to record briefly the results of an extensive search of medical and surgical works, as well as the records of individual cases, in the hope that by grouping together many isolated instances I may be able to contribute somewhat to our exact knowledge of the surgical complications and sequels of the continued fevers, both as to their causes, the means of their early recognition, the best methods of treatment, and, if possible, that still higher object—the averting of dangers which otherwise may prove disastrous to health, and too often to life itself.

The exanthematous fevers are better known as causes of surgical troubles, and I have therefore carefully excluded them, as well as a few cases following intermittent fevers.

I shall omit entirely all well-known results of a semi-surgical character, such as hemorrhage from the nose and bowels; peritonitis, with or without perforation; erysipelas, a not infrequent complication about the face or when bed-sores exist; and cases of thrombosis of the veins, which cause a variety of phlegmasia deserving a more extended study than it has yet received. Bedsores and the ordinary abscesses are too well known to demand other than passing allusions. Much as I regret to do so, I must also omit, from want of time, the consideration of the forms of disease which especially interest the ophthalmic surgeon. Ulceration and perforation of the cornea are briefly, but I may say completely, treated by Trousseau.[1] Post-febrile ophthalmia, or amaurosis,—a peculiar retino-choroiditis which follows only relapsing fever, first described by Hewson, in 1814, and by Wallace in 1827,—has been so carefully studied of late by ophthalmic writers as to leave but little further to be said.

I shall include only such cases as diseases of the joints; edema glottidis and necrosis of the cartilages of the larynx, which often require tracheotomy; necrosis of the bones; gangrene of the extremities and other parts; fistulæ of various kinds, and the like.

The records of many cases are extremely imperfect; some, in fact, are mere allusions, and I have been compelled, there-

[1] Clinique Méd. de l'Hôtel-Dieu, 2d ed., p. 271, and Gaz. des Hôp., 1856, 170.

fore, in tabulating them, to come as near the truth as may be. The frequency of many symptoms is therefore greater than appears from my tables. Especially is it difficult to discriminate between typhus and typhoid fevers as causes. The earlier cases, before the essential abdominal lesion of typhoid was recognized, are all classed as typhus, and even to-day many cases, especially in German books and journals, are briefly called "typhus," meaning "typhus abdominalis"—*i. e.*, typhoid. If any error exist, therefore, it will consist in assigning too many cases to typhus proper, for I did not feel permitted to go back of the record unless plainly authorized to do so by the history or the post-mortem.

I. Diseases of the Joints.

Two forms of disease of the joints are found: first, a polyarticular inflammation, which may assume either a rheumatic or a pyemic form; and, secondly, a monarticular inflammation.

The rheumatic variety I shall at once dismiss. The pyemic form of inflammation is not very common, for Murchison, with his immense experience in the London Fever Hospital, has seen but one case. It follows the usual course of pyemia, both in its symptoms and its usually fatal issue. Huss and others have referred it to suppurative phlebitis from bedsores, parotitis, etc., but it has been observed in cases in which no such complication existed. Pyemic arthritis, like the gangrene from pressure, parotitis, etc., is most apt to occur in severe cases, in which the blood change is at its maximum, and the "typhous crasis," as Stokes has expressed it, possibly becomes converted into pyemia.

It is, however, the monarticular form which will most interest us. It affects the larger joints, such as the elbow and shoulder, the ankle and knee, but above all the hip. The pain is usually slight. The swelling is generally readily observed in all joints except the hip and the shoulder, where it is probably obscured by the muscular masses about these joints combined with the tardy increase in the swelling. Usually it arises spontaneously, but occasionally from periostitis or necrosis extending into the joint. It rarely produces suppurative or fistulous openings. The

result is, therefore, generally a gradual return to usefulness, although in three cases I have found ankylosis. Of 43 cases, the lower extremities were affected in 39, the upper in only seven, three of the cases involving a joint in both, for occasionally two large joints are affected at once. Arthritis, therefore, resembles other surgical febrile affections, such as gangrene, necrosis, etc., in affecting mainly the lower extremities, as do also thrombosis and the ordinary edema. The frequency of these joint troubles is not great. According to Güterbock,[1] in a series of years in the Charité (Berlin) and the Hamburg Hospitals, not a case occurred, and in the Vienna General Hospital, from 1868 to 1871, only two cases among 3130. Murchison does not even name this complication at all, nor do any other of our text-books, either on surgery or practice, except a few lines by Volkmann in Pitha and Billroth's Handbuch. Güterbock and Hellwig[2] are the only authors who have treated them at all fully. Yet that they are of great importance, and demand our utmost attention, will be seen at once when we consider that of the 43 cases named, spontaneous dislocations occurred 27 times in the hip, twice in the shoulder, and once in the knee.

These dislocations require more particular notice. From their similarity to febrile arthritis in the same and other joints their pathology seems clear, although—in singular contrast to the strangely fatal laryngeal stenosis I shall soon consider—not a single death has occurred, and therefore no post-mortem verification has been possible. They belong to the class of "distention luxations." That the cause is not the specific poison is evident, since similar results follow other and dissimilar diseases, such as locomotor ataxia, the exanthematous fevers, hemiplegia, sciatica, and rheumatism, as pointed out by Stanley in 1841.[3]

Usually, in the period of convalescence, following therefore the prolonged exhaustion, there arises a subacute synovitis, with a gradual serous distention of the capsular ligament, which, having reached a certain point, may slowly

[1] Ueber spontan. Luxat. und einige ander. Gelenkkrankh. bei Ileotyphus, Langenbeck's Archiv, xvi, 58.

[2] Ueber die Affect. des Hüftgelenk. bei Typhus, Marburg, 1850.

[3] On Dislocation, Espec. of the Hip-joint. Med.-Chir. Trans., xxiv, 123. See also Malgaigne, Fract. and Disloc., Paris, 1855, ii, pp. 218–226, 882–887.

subside, and no further evil follow. In three cases, however, this burst externally, producing sinuses, but in none of them was the discharge purulent. The main result is a slow, generally unperceived, elongation of the ligaments,—*e. g.*, of the hip,—with perhaps also a swelling of the so-called gland at the bottom of the acetabulum. This distention will spend its force mainly posteriorly, since the inverted Y-ligament reinforces the capsular ligament in front. Given this condition, the slightest force will dislocate the head of the femur upward and backward on the dorsum of the ilium. In one case a fall to the floor produced it, in three others turning over in bed, and twice the lifting of the patient in the arms from one bed to another. But in all the other 21 cases no cause was assignable, and it is, therefore, likely that it was mere muscular contraction which becomes, at the time when these occur, more vigorous as health gradually returns. Seitz has recorded one of the most remarkable cases, in which, from extensive bedsores, the abdominal decubitus was maintained for nearly a month, and he supposes that this was the immediate cause of the dislocation. But as in no other case is this posture noted, it can not be regarded as correct. Indeed, if posture have any influence, as the dislocation is generally if not always iliac the dorsal decubitus would be the most favorable for its production. In one of the shoulder cases a subcoracoid luxation was caused by the patient's assuming the erect posture. Gravity had here probably some influence, together with the muscular exertion. The dislocation of the knee also was posterior.

A remarkable case, corroborative of the non-specific character of the lesion and the probable influence of gravity, I have lately seen in the service of Dr. Wm. G. Porter, at St. Mary's Hospital. The child was about two years of age, greatly exhausted from malnutrition, and for about six weeks was kept alive by inunctions of sweet oil, no other nourishment whatever being given. It had large abscesses in different parts of the body, and at present has necrosis of the left humerus. Early in the period of returning strength, and before the necrosis appeared, spontaneous luxation of the left humerus into the axilla occurred. It was easily reduced by manipulation, and has not since recurred.

Typhoid was noted as the preceding fever in 15 and typhus in 7 of the hip cases. Sex has a marked predisposing influence in this, as we shall find in other diseases, for of 23 cases, 15 were males and only 8 females. The age at which they occur is still more noteworthy: 15 were under fifteen years, 6 from fifteen to twenty, 1 was thirty, and one was sixty-one years old; that is, 21 out of 23 were under twenty years old. The analogy to coxalgia, it will be observed, is therefore very marked. Usually they were single dislocations, 6 being on the right side and 6 on the left; but in 3 cases dislocation of *both* hips occurred.

From the apathetic condition of the patient in some cases, the subacute nature of the lesion, the absence or slightness of the pain, the masking of the swelling by even the wasted muscles about the joint, and, above all, the want of knowledge of cause or probability of the dislocation, and therefore the neglect to examine the parts thoroughly, it is not surprising that this threatening evil should have been often unobserved. In 9—that is, one-third—of the cases it is distinctly stated that the *actual dislocation* was the first fact observed, and in most of the others this is probably true.

The date at which the dislocation was, at least, observed, was generally after the third week. One case occurred in the first week, 4 in the second, and 9 in the fourth week or later—that is, during distinct convalescence. Pain was experienced in 13 cases. Usually it was not severe, nor was it always strictly localized in the hip, but sometimes extended to the entire leg. In only two cases was it referred to the knee, thus differing markedly from the well-known coxalgic knee pain. Swelling is only distinctly stated in 6 cases, though probably present here as in other joints, but either unobserved or often unrecorded in the brief statements I have often found. The variety of the dislocation is not named in 10, but as in all the other 17 it was iliac, there is good reason to believe that this is probably always the case. Shortening is recorded in 11 cases, and where the amount is named was generally 1½ to 2 inches. In 5 cases the rotation was inward, in 2 outward, and in 2 of the 3 double dislocations both legs were rotated in the same direction,—that is, right or left,—thus producing a peculiar deformity when compared with the apparently

reversely rotated body. The head of the bone in 4 cases was freely movable in all directions. This mobility of the head and the singular diversity in the rotation of the limb are additional reasons in favor of the distention theory of its pathology. Flexion and adduction Dr. Sayre has shown to be the position of the limb which produces the greatest capacity of the capsular ligament of the hip, and we ought to see this position, therefore, as a rule, in distention luxations. But I only find two cases in which there were adduction and flexion. In the other cases the position is not stated, except in one in which the limb was extended.

As to treatment, reduction is generally easy when the luxation is discovered early, but if the discovery or treatment be tardy it is always difficult and often impossible. In 11 cases reduction was successfully accomplished—seven times by manipulation, twice by extension, and twice by both means. In 8 cases reduction was not effected, and in 8 the result is not stated. Only 2 cases of recurrence of the luxation are noted, a rather surprising fact in view of the relaxation of the distended tissues; but its possibility should be borne in mind and guarded against by the same prophylactic means that I will name directly. No snap is heard on reduction, all tension and suction-power of the joint being lost. Even after reduction the leg may be somewhat longer than the other, owing, probably, to the distention, to the swollen articular gland, and possibly in old cases to interstitial changes in the neck of the femur.

The question of prophylaxis is perhaps the most important of all, and the indications are clear. First, a careful watching and repeated examination of the hip-joint, especially in children, to detect any effusion. If any exist, the position of the leg becomes of the greatest possible importance. As adduction and internal rotation favor spontaneous dislocation, the leg should be kept in abduction and external rotation. The first indication is easily fulfilled by two lateral sandbags, which may be bridged across in front at intervals by a bandage, to keep the leg at rest between them, or by lateral splints. The foot may be kept in external rotation by bandages or adhesive plaster fastened to the external sandbag or splint. If the effusion threaten to produce dislocation, it may well be a question whether aspiration would not afford a safe and efficient means of prophylaxis.

II. Diseases of the Bones.

A popular name for necrosis is "fever sore," but, as Nathan Smith long since pointed out, more because it caused fever than because it was caused by fever. That it does follow fever and is caused by it is certainly true, but it is not a very frequent though a very important sequel. I have collected thus far 50 cases of necrosis proper following continued fevers, but among these are 19 reported by one single author,—Whately,—the histories of which are exceedingly brief and unsatisfactory. He states, indeed, that he has *seen* 30 cases—an incredible statement, I think, in view of the fact that from all other sources, after an extended search, I can only gather 31 more. "Fever" with him, however, may include a very wide range. One element of unavoidable uncertainty in the history is seen at once. The osseous disease usually falls under the eye of the surgeon at a period distinctly subsequent to the fever, and, knowing nothing personally as to the previous medical history, he must depend upon the statement of the patient—often a most unreliable means of information.

Two causes for such necroses and other forms of disease, such as periostitis and caries, are to be found: first, thrombosis, or in some cases possibly embolism; and, secondly, absolute inanition or want of nutrition.

The rôle assigned of late to the marrow together with the spleen as a source of the red corpuscles, would seem to be confirmed by the similarity of the changes observed by Ponfick [1] and others in the spleen and marrow in typhoid. In later convalescence, or shortly after recovery, we find in the marrow many mother-cells holding numerous blood-cells, enormous masses of large cells filled with pigment in complete analogy with the observed metamorphoses of extravasated blood. These are especially seen at the sides of the cavernous veins, and must retard still further a circulation already impaired in force by a weakened heart. Nutrition is here at its lowest ebb, and as the vessels, from the nature of the tissue in which they run, can not enlarge in proportion to the needs of the circulation, and are them-

[1] Ueber die sympathischen Krankh. des Knochenmarks bei inneren Krankh., Virchow's Archiv, lvi, 524. *Cf.* also Anatom. Studien über den Feb. Recurrens, Virchow's Archiv, 1874, lx, 153.

selves more or less involved in fatty degeneration, we may readily understand how the lack of nutrition alone, as in Dr. Porter's case previously cited, would cause gangrene of the bone even more readily than in the soft parts, in which we know it to be so common.

That the bones should suffer from vascular clots, and especially the bones of the lower extremities, where such clots are most frequent, as we shall see, in gangrene, is probable both from analogy and experiment, and from one case in which it has been actually observed in typhoid.[1] There is no reason to suppose, when thrombosis is so frequent elsewhere, that the bones would escape. Virchow has shown that in relapsing fever we frequently have infarcts in the marrow. Volkmann[2] gives an excellent case and illustration of necrosis of the tibia and talus from embolism, the result of endocarditis. We need a few similarly exact observations in cases of necrosis from fever, in which death or amputation affords the desired opportunity to settle the question positively; but generally the examination, if made at all, is of the most superficial character. Hartmann[3] has shown, experimentally, that obliteration of the nutritious artery causes necrosis of the inner lamella of bone—a strong point it must be admitted, in favor of Whately's theory that after fever the result is not ordinary necrosis, but a central necrosis of the inner lamella which he limits to the tibia. Blocking of the veins is evidently not so dangerous in bones as blocking of the arteries, since the collateral venous circulation, especially toward the extremities, is abundant, while the collateral arterial circulation is scanty.

I have found 69 cases of diseases of bone following continued fevers. Of these, 50 were cases of necrosis, 12 of caries, 3 of periostitis, and 4 of indeterminate or doubtful nature. Three cases of necrosis following typhoid combined with small-pox I have excluded. Typhoid, as usual, claims the larger share; for of 41 cases, 37 followed typhoid and

[1] See Meusel's case, p. 344.

[2] Pitha and Billroth's Handbuch, Bd. ii, Abth. i, Lief. i, p. 287; and Langenbeck's Archiv, 1864, v, 330. See also Mollière, Lyon Méd., 1870, pp. 12, 149, 256; 1871, p. 38.

[3] Nekrose herbeigeführt durch Verstopfung des Foram. nutrit., Virch. Archiv, viii, 114.

only 4 followed typhus. Males also are in the preponderance, counting 38 to 14 females. Age has not a very marked influence, as 19 were under twenty years, 11 from twenty to thirty, 11 from thirty to forty, and 5 over forty. Scarcely any region of the body escapes: 22 cases involved the head, 7 the trunk, 6 the upper extremities, and 42 the lower, a result strikingly in accord with the cases of arthritis and gangrene. In the head I have found 12 cases of necrosis of the alveoli and jaws. Among these perhaps the most remarkable, although somewhat doubtful, case is the one I saw in a soldier at Frederick, Maryland, in 1862, in which, after typhoid fever followed by pneumonia, the entire right upper jaw with a part of the palate bone and the intermaxillary bone necrosed and separated. The case is remarkable, both from its being a striking example of the limitation of disease by the embryonic development,[1] and also from the extraordinary series of operations subsequently done by Dr. Gurdon Buck, of New York,[2] to remedy the frightful deformity which had been produced. It is but proper to say that the man was reported to have taken about two drams of various mercurials during his preceding illness; but from the facts I have stated, as well as his scanty history, I think it tolerably clear that the fever, and not the mercury, caused the necrosis. Mercury or syphilis complicated two or three of the other cases I have tabulated, but they were not, apparently at least, the cause of the trouble.

Mr. Salter[3] has pointed out the relation of alveolar necrosis to the eruptive fevers, especially scarlet fever, and believes that as these structures are dermal in character they partake with the skin in the eruptive mischief. While this relation remains undisturbed, yet I do not think the necrosis exists as a specific sequel of these fevers only. Of the 12 cases cited, 7 occurred as follows: 1 at sixteen, 1 at twelve, and 5 at ten years of age and under—that is, during the period of dental development and

[1] H. Allen, Studies in the Facial Region, Phila., 1875, has specially called attention to this point.

[2] Trans. N. Y. State Med. Soc., 1864, p. 173; Circ. No. 6, S. G. O. Surg. Sec., Spec. 557, p. 53; Med. and Surg. Hist. War of the Rebel., Pt. i, Surg. vol., pp. 375–377, and Buck on Reparative Surgery.

[3] Holmes' Syst. Surgery, 1st ed., vol. iv, p. 50.

growth. That such cases are more frequent in the exanthemata is natural when we consider the relative infrequency of the continued fevers under fifteen years of age.

The period at which these diseases of the bones arise varies greatly. Of 47 cases, 10 arose in the first two weeks, 27 in from three to six weeks, and the remaining 10 followed often months after the fever. The earlier cases include, probably, most of those from clots, and the later ones those arising from enfeebled nutrition, whose effects, especially in structures which vary so slowly as the bones, may readily extend over such long periods.

Especially does this enfeebled nutrition show itself in cases where too early strain is put upon the parts and justifies the remark of Aitken that "no man can be considered fit for work or for general military service for three or four months after an attack of severe typhoid fever." The following case illustrates the wide-spread mischief that may follow in the osseous system when put to the test by labor, months and even years after such a fever.

[For the full history of this case, see Case IX, H. W., p. 123.]

The symptoms need scarcely be alluded to, for they are those of ordinary necrosis, although Whately endeavors to differentiate them. In 13 cases of necrosis of the long bones other than Whately's in which the description enables me to judge, I find only 3 distinct cases of central necrosis,[1] and these differ in no especial manner from other cases. That it is limited to the tibia, as asserted by Whately, is disproved by the fact that of 77 bones affected, the tibia was attacked only 30 times, including in these the 19 reported by Whately.

The results of necrosis vary with the situation. The

[1] The third of these cases I have had in private practice while the MS. is passing through the press. A. W., a rather feeble girl, age eleven, was taken sick with typhoid May 10, 1876. After three to four weeks in bed she began to walk, but soon had to stop on account of weakness, and especially of pain in her left tibia. After three weeks' poulticing it broke in two places, and has discharged ever since. I saw her first in December, 1876, and found two small sinuses which extended into the bone, but no dead bone had ever been discharged. After building up her general health by tonics and cod-liver oil, on February 17, 1877, I operated on the bone, using Esmarch's apparatus in the manner I have suggested (Phila. Med. Times, Sept. 26, 1874), and after making an opening into the medullary canal with the chisel and gouge, I removed a small, loose spicula of necrosed bone (central necrosis) ⅞ of an inch long. At this date, March 5, 1877, she is doing well.

ordinary sinuses, etc., I need not mention further. If in the sacrum, coccyx, or innominate bone, perineal fistulæ may result, of which I have found three cases. If in the mastoid or petrous bone, the brain and its membranes may be involved. The following résumé of the case of Meusel is of especial interest, as it throws so much light on the cause of the necrosis, the clot in the meningea magna, and is as extraordinary for the audacity of the treatment as for the success of the result:

A student in the gymnasium, age nineteen, at Easter, in

Fig. 1.—Necrosis of frontal (A), parietal (B), and greater wing of sphenoid (C) bones, following typhoid. In B and C the grooves for the middle meningeal artery and its branches are seen. Natural size. (Meusel, Deutsche Klinik, 1872, page 266.)

1868, had an attack of typhoid fever, went home when convalescent, but did not improve, and suffered much from headache. In August, four months after the fever, he had a large fluctuating abscess over the right parietal region, which was opened, and dead bone found. By October 1st the bone was loose, and on the 5th an incision of three inches was made and a loose piece of the frontal was removed. The rest of the dead bone was firm, but the

incision was extended backward until the whole of the necrosed portion was exposed. It was then carefully chiseled loose and separated at the squamous suture. At the anterior inferior angle the necrosis was there found to extend on the internal surface only; with a fine chisel this internal lamella, a piece 1½ × 2 cm., was chiseled away from the great wing of the sphenoid. In it was a groove in which lay the anterior branch of the middle meningeal, filled with the detritus of a clot. The whole piece was 5½ × 9 cm. The dura mater was but slightly injected. The scalp and the dura mater united, and in fourteen days he was nearly well, having recommenced his Latin and Greek with the greatest zest eight days after the operation. Two small pieces of loose bone afterward caused threatening symptoms, but improvement followed immediately upon their discharge. In March he was entirely well, and went to Göttingen to study philology at the University. Epileptiform attacks followed during 1869, but then disappeared, and had not reappeared in 1872.

As to treatment, the ordinary operation for the removal of necrosed bone is to be done at the proper time, especial care being taken to remove any small central sequestrum. Occasionally the disease of the bone may cause extensive disease in the soft parts, or may extend to a neighboring joint, though either complication is rare. Amputation then becomes imperative. Only four such amputations occurred in the cases reported; two died, one recovered, and one was under treatment. About the face not infrequently extensive plastic operations are required.

III. Diseases of the Larynx.

The laryngeal complications are noted very briefly by several medical writers, such as Murchison, Flint, Liebermeister, Griesinger, etc., but it is mainly the laryngeal ulcers themselves which are treated of, their surgical results being scarcely mentioned. Gross and Gray barely allude to typhoid as a cause of edema glottidis. Even systematic writers on the larynx scarcely notice them. Gibb [1] and Rühle [2] refer to two or three cases. Cohen

[1] Dis. of the Throat and Windpipe, 2d ed., 292–294.
[2] Die Kehlkopfskrankh., Berlin, 1861, 157, 257, and Fig. 2.

simply names fever. Türck [1] gives, however, eight valuable cases. I have collected 169 cases, of which at least 67 (and probably many more) certainly involved the cartilages themselves.

The troubles which may demand surgical interference are all allied, and are the result of a low grade of inflammation. The entire respiratory mucous membrane (as is shown by the frequency of bronchitis) is in a more or less catarrhal condition, like that of the bowels, and occasionally other mucous membranes, such as those of the gall-bladder, urinary bladder, and vagina. It is not, therefore, a matter of surprise that serious trouble should arise in the larynx, especially as slight variations in its mechanical condition gravely embarrass so vital a function as respiration.

Pathologically, the troubles may be grouped into three varieties—viz.: (1) Edematous laryngitis; (2) ulcerative laryngitis; (3) laryngeal perichondritis. Practically it is often exceedingly difficult to separate these various forms, even at the post-mortem, so far do they overlap each other. Edema may exist alone or it may result from either of the others; ulceration may march steadily deeper until the cartilages are involved; or the perichondritis may produce an abscess which will burst, and so form an ulcer. How much more difficult, nay often impossible, then, is it to diagnosticate precisely the form of the disease, when, happily, the patient recovers. Dyspnea, suffocation,—this is the one great overshadowing clinical fact which groups them all together, whatever the form of the disease or of the preceding fever.

That simple asthenic edema may arise just as edema of the lower extremities is not only probable but has been positively observed by Emmet [2] and Buck.[3] It has also been observed after diarrhea, bronchitis, and other diseases; but this is a much rarer form than those cases in which it is secondary to erysipelas, or parotitis, or laryngeal ulcers, often of small extent. I can not help suspecting, also, very strongly that more careful future examinations will

[1] Klinik d. Kehlkopf., Wien, 1866, 215–235.

[2] Amer. Jour. Med. Sci., July, 1856, xxxii, 63.

[3] Trans. Amer. Med. Assoc., 1848, 135.

show in not a few cases that local venous thrombosis has been the cause of the edema.

The other two forms especially merge into each other. Rokitansky believes that the ulcers are a peculiar form of typhus—the so-called laryngo-typhus. Others, and I certainly agree with them, do not believe that they are specific in their origin, but belong "to the common cortège of septic diseases" and other allied disorders in which the low grade of inflammation readily runs into ulceration, and even into local gangrene. How much influence local stasis of the blood or even clots in the vessels may have has not been carefully investigated, but I believe them to be no unimportant factors.

These ulcers are sometimes very common. Thus, Griesinger [1] met with them in 31 out of 118 autopsies, Hoffmann [2] in 28 out of 250, and Louis believes that "if found on the body of one who has died of an acute disease, they will establish with nearly perfect certainty, and without going any further, that the affection is typhoid fever." At other times they are so rare that in nearly 13,000 typhus cases at the London Fever Hospital, Murchison records but 21 of laryngitis, of whom 8 died; and in typhoid he has only seen three or four cases.

Where the cartilages are involved, Moritz-Haller [3] and others believe that it follows the ulceration which destroys the mucous membrane and eats down to the cartilages, while Sestier [4] has gone so far as to declare that the ulcers which accompany perichondritis are not primary but in all cases secondary. Both, I believe, are right, but both go too far. So far as the history and post-mortem appearances would enable me to judge, I have found in 20 cases that the perichondritis preceded the ulcers and caused them, while in 10 cases the ulceration had caused the perichondritis. In cases of perichondritis in which death takes place early, there is no opening in the mucous membrane, but a submucous abscess will be found surrounding the necrosed cartilage. If death takes place at a later date, a small opening will exist, through which the probe

[1] Infectionskrankh., 1857. Bd. ii, Abth. ii, s. 160.

[2] Veränd. d. Organ. beim Abdom. Typhus.

[3] Oesterr. Zeitschr. prakt. Heilkunde, 1856, No. 19.

[4] Arch. Gén., 1850, 4me sér. xxxiii, 385; xxiv, 35, 297, 441.

will enter into a much larger cavity. In other cases the surface mischief will be by far the most widely spread, the ulcers being roughly conical, involving not only the mucous membrane, but eating deeply down to the cartilages. Similar necrosis of the nasal cartilages also sometimes results from fever.

Those cases in which there is considerable cough, or the patient in his delirium has cried aloud, or sung much, or those in which, after distinct convalescence, there has been exposure to wet and cold, are predisposed to laryngeal troubles. They are exceedingly rare in children. In 94 cases in which the age is recorded, I have found but 6 under fifteen years of age, 60 from fifteen to twenty-five, and 28 above twenty-five years. Sex is potent here as in the other diseases considered. Lisfranc thought them more common in women than in men, but of 110 cases, I find 86 in men and only 24 in women, or 3½ to 1.

The cause of the stenosis is various. It may be: (1) from edema; (2) the swelling produced by the abscess about the cartilage; (3) the sides of the glottis may fall together if the cricoid be destroyed and in pieces; (4) the permanent approximation of one or, more rarely, of both, vocal chords from destruction of the fixed points of origin of the muscles; (5) as in two remarkable cases given by Hoffmann,[1] shreds of sloughing tissue, on which blood coagulates, may form a sort of polyp, which suffocates the patient even in spite of tracheotomy (Fig. 2).

The seat of the stenosis is threefold. Most frequently (25 cases) it is supraglottic—that is, in the epiglottis and ary-epiglottidean folds, especially where the edema is primary, or where it is caused by ulceration or arytenoid perichondritis. The next most frequent site is subglottic—*i. e.*, about the cricoid (22 cases). This is always, I believe, the result of cricoid necrosis or perichondritis. Russell[2] reports two cases following typhus, which he regards as examples of Gibb's "subglottic edema." That the second was a case of ulceration and perichondritis is quite certain from the history, and most likely the other was too. The larynx was normal down to the chords, tracheotomy rescued both when suffocation was imminent, and both were

[1] *Op. cit.*, pp. 253 and 255.
[2] Glasgow Med. Jour., Feb., 1871, 209.

followed by stricture, requiring the permanent use of the cannula. The least frequent site of the edema is in the glottis proper, since edema of the vocal chords is named but nine times.

The date of the development is generally here, too, in the later fever, or more frequently in distinct convalescence. Of 102 cases only 4 occurred in the first week, 13 in the second, 19 in the third, and 66 from four weeks to two months. They follow typhoid far more frequently than typhus, in the proportion of 106 to 49, some of the latter

Fig. 2.—Laryngeal ulcer after typhoid. Polypoid hematoma hanging from it and causing death by suffocation after tracheotomy. *a*, upper end of incision. The smaller cut, *b*, is a section of the hematoma showing its two layers, the center consisting of shreds of dead tissue hanging from the ulcer, and the outer layer of clotted blood. (Hoffmann, Veränd. d. Organ. beim Abdom. Typhus, Tafel v, Fig. 16.)

being probably really typhoid, while 14 arose from other forms of continued fever.

The position of the ulcers in the larynx is noteworthy. Wherever they may be, from the arytenoid to the cricoid, they are almost invariably posterior. Rheiner [1] has shown that the posterior wall of the larynx is the richest in vessels, and that ossification begins here often as early as the twentieth year. Here, then, we should expect the most frequent inflammations and thrombosis of the smaller ves-

[1] Beiträge zur Histologie des Kehlkopfs; Würzberg, 1852.

sels ; and when we add to this the effect of gravity, from the continuous dorsal position, and the mechanical effects of frequent use of the voice, and, therefore, repeated movement of the arytenoid cartilages in some delirious cases, we have a sufficient explanation of the phenomenon. Emphysema of the neck and trunk is an occasional result of such ulcers where they penetrate the mucous membrane. This was first pointed out by Wilks.[1] Other cases are reported by Steiner[2] and Loeschner.[3] All three were children.

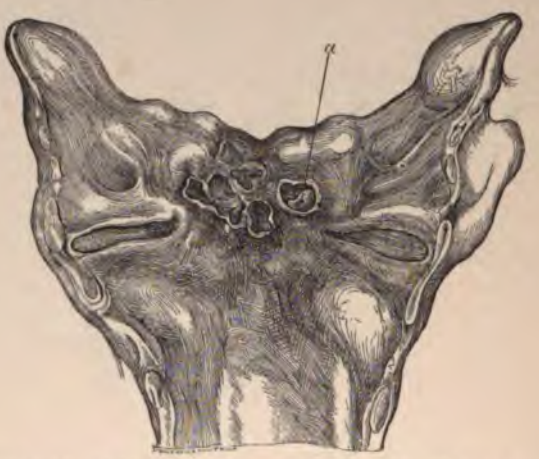

Fig. 3.—Perichondritis laryngea following typhoid. Ulcers on the posterior wall of the larynx. At *a*, a piece of the necrosed left arytenoid cartilage shows. (Türck, Krankh. des Kehlkopfes, p. 216, Fig. 78.)

Necrosis of the cartilages is the most important form to recognize, in consequence of its excessive gravity ; for, of 56 cases in which the result is given, 54 died. One recovered after tracheotomy,[4] and one without it.[5] We can scarcely agree with Türck, therefore, that the prognosis is "doubtful." The seat of the necrosis in the majority of the cases is the cricoid (38 times), next, the arytenoids (19

[1] Med. Times and Gaz., 1862, ii, 276, and Trans. Path. Soc., London, 1857, ix, 34.

[2] Dis. of Children, 1874, 363.

[3] Prager Vierteljahr., 1856, iv, 23.

[4] Türck, p. 223.

[5] Hérard, l'Union Méd., July 14, 1859, quoted by Trousseau, Clin. Med., Syd. Soc. Trans., 2d ed., vol. ii, p. 407.

times), while the other cartilages were affected but 5 times. In 10 of the cases the cricoid and arytenoid were involved simultaneously.

It is to be specially observed that probably a number of the cases which recovered were also really cases of perichondritis, of which the *positive* evidence was wanting.

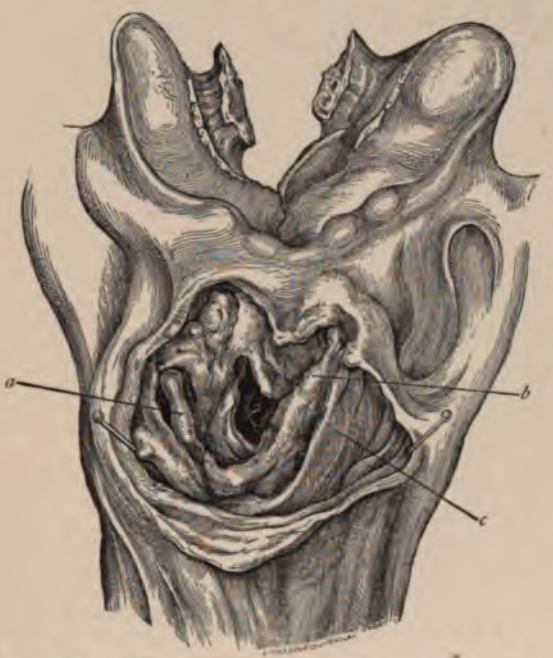

Fig. 4.—Perichondritis larynges following typhoid; abscess opened from esophagus and seen from behind: *a*, *b*, Necrosed and partly destroyed cricoid cartilage; between *a* and *b* the dark area indicates the point where the abscess communicated with the trachea; it did not communicate with the esophagus; *c*, crico-arytenoideus posticus muscle. (Türck, Krankh. des Kehlkopfes, page 218, Fig. 80.)

Thus, in 8 cases in which recovery followed tracheotomy, the patients spat up gangrenous or purulent matter, besides having other symptoms of cricoid necrosis; but as the expectoration of any pieces of necrosed cartilage was not positively observed, I have not included them in my statistics of necrosis. Were they included, the result, and especially the result after tracheotomy, would be far more favorable.

that both by direct and lateral pressure on the cricoid, and also by sliding it sidewise, we may often elicit pain which might otherwise be overlooked. Lateral pressure will also often produce dyspnea, or aggravate it, especially if the cricoid be broken, or nearly broken, in two. Dysphagia is noted as present 21 times and absent 5 times, and is especially present in cricoid and arytenoid necrosis. Rarely is there any external swelling to attract attention, for I have only found it noted thrice. Nor can the pharynx be relied upon to warn us of impending evil, for in 16 cases in which its condition was observed, it was normal in 10 and inflamed in only 6.

Most of the cases occurred before the days of the laryngoscope, so that I have only 13 such examinations from which to draw any inferences, 7 of which are recorded by Türck. In several instances there was nothing whatever abnormal down to the vocal chords ; and the swelling of a cricoid abscess on the posterior wall is not visible. But the usual facts observed were the fixation of at least one chord in the middle line, diminished mobility of the other, swelling of the ary-epiglottidean folds, stenosis of the larynx increased by lateral pressure, a depression of the mucous membrane in case of destruction of the arytenoid, and sometimes the opening of an abscess, usually near the processus vocalis. A few such positive facts with any additional positive symptoms should lead to an equally positive treatment.

And first the mortality is so great that the treatment is of the greatest possible moment. Of 146 cases of all kinds of stenosis in which the result is recorded, 101 died and 45 recovered, a mortality of over 69 per cent., but not a surprising result when we add the laryngeal disease to the exhausting fever. But when we separate the cases operated upon from those in which no operation is named, or was certainly not done, the importance of the treatment becomes even more appreciable. Of the 76 cases not operated on, in which the result is stated,—and, be it observed, I have included in these the cases of mere scarification,—17 recovered and 59 died, a mortality of over 77 per cent. Of the 70 cases operated on by some form of bronchotomy, 28 recovered and 42 died, a mortality of 60 per cent. And when it is remembered that in two of the

fatal cases the larynx was not opened, though tracheotomy was apparently performed; and a third, in full recovery thirteen days after the operation, on the removal of the cannula was suddenly suffocated before it could be replaced; and in another the cannula got displaced in front of the trachea; that in many, if not in most, of the cases, the operation was deferred until the last possible—that is, the most unfavorable—moment; that many cases that might have been rescued were plainly *allowed* to die from exhaustion, or even from positive suffocation, by timid doctors, in which the result could not have been worse had an operation been performed, the question of operation would seem to be decided.

Yet I would not be understood as an advocate of rash and indiscriminate bronchotomy. Its dangers are great, and not to be undervalued. The question, however, is often between a dangerous operation and a more dangerous refusal. In case the attack is sudden and severe, so that life is in immediate and positive peril, no question can arise as to the propriety of an operation, wanting which the patient perishes on the spot; nor any question that crico-thyroid laryngotomy is the easiest, safest, speediest operation. Delay here means death. Of 14 such operations, 8, or 57 per cent., recovered, and in such cases as are recorded by Emmet and Anderson[1] no words can add force to the fact that after life was apparently extinct, laryngotomy and artificial respiration saved five, the delay of a few minutes resulting fatally in the sixth case. A cut throat here is not over-dangerous, and the operation is so simple and may be so imperative that every medical man, as well as surgeon, should stand ready to do it in case of impending death.

But it is in the less suddenly threatening cases that judgments may differ, and here I hope to be able to assist in forming a decision. The moment, in a case of typhus or typhoid, that hoarseness, aphonia, dyspnea, or dysphagia sets in, the larynx should be examined with the utmost vigilance both from without and by the laryngoscope. If edema or ulceration be found, but the danger be not as yet severe, leeches, iodin, ice in rubber bags, or, possibly, a blis-

[1] Ten Lectures on Fever, London, 1861, 46.

ter may be used externally, and astringents, nitrate of silver, etc., internally, together with proper general treatment. In 1819 Lisfranc proposed scarification and operated successfully in six non-febrile cases, and in 1847 Buck revived the practice and devised an appropriate knife. The laryngoscope has made such treatment far more certain, and it should first be tried in suitable cases.

But, should these means fail? Then an *early* rather than a late tracheotomy, for otherwise we add to the previous enfeebling disease a prolonged battle for breath, with its ensuing pulmonary congestion and general exhaustion. Especially is this to be commended in the peculiarly fatal necrosis of the cartilages. Of 67 positive cases, 41 of which occurred in men and only 10 in women, all died but 2, one with and one without an operation; 22 were operated on, 45 were not. But, as before mentioned, 2 died from displacement of the cannula, at least one of which had practically recovered; 8 cases of recovery after tracheotomy are not included, since, though the presumption in favor of necrosis is very strong, yet the evidence is not positive. Were only these 8 included, and it is certainly fair to do so, it would stand 30 operations and 10 recoveries, a mortality of 67 per cent., and 45 not operated and 1 recovery, a mortality of nearly 98 per cent.

Once, then, that perichondritis is established, death is almost unavoidable if no operation be done. If bronchotomy be resorted to, the chances are greatly improved. Indeed, it is probable that if the dangers which surround such cases be recognized in the future, and a prompt attempt at relief be made, far more favorable results will be obtained. I would, therefore, join with Sestier in urging an immediate operation the moment that perichondritis is recognized and serious dyspnea sets in, without waiting for repeated attacks to exhaust the slight store of strength, all of which will be needed during the subsequent separation of the necrosed cartilage, and the sloughing of the soft parts.

In other cases than those of perichondritis such haste will not be necessary: yet if the respiratory murmur be progressively enfeebled; if pulmonary congestion set in; if the paroxysms of dyspnea increase in frequency and severity, especially if orthopnea arise; if the local disease

be extensive or rapidly increasing; or if the general feebleness be so great that a little further interference with the respiration will destroy life; then no time is to be lost. Doubt is certain death.

What operation shall be performed? If an instant operation be needful, crico-thyroid laryngotomy is the best; if time allow, tracheotomy. But if the cricoid be involved, with Beck,[1] I would advise laryngo-tracheotomy,—*i. e.*, tracheotomy prolonged through the cricoid,—since it would allow readier access to the seat of the disease for the discharge of the pus, the removal of any loose piece of cartilage, and the treatment of any ulcers or granulations. These I regard as greater advantages than the danger of possible collapse of the lateral halves of the cricoid, which is prevented in part by the cannula, and would not impede the respiration even if it occurred. Unless necessary, it is best not to operate during a paroxysm; since the mechanical difficulties of the operation are then largely increased, and the danger of entrance of air into the veins is apparently much greater. In 36 operations for laryngeal angina, two such accidents occurred; whereas, in 245 non-anginose cases, not a single similar accident arose (Sestier).

Hemorrhage, as would be supposed from the condition of the blood, is sometimes a serious complication, both at the operation and subsequently, and caused death in three cases.

A curious and unexpected complication arose in two cases reported by Mohr[2] and Laennec,[3] the knowledge of which should guard us against a similar error. The operation having been apparently achieved and the cannula inserted, respiration was not bettered nor did air pass through the cannula. In a few minutes the patients died suffocated. At the post-mortem it was found that the cannula had entered, not the larynx, but the abscess around the cricoid. In Mohr's case the vertebræ were felt through the incision, and a probe and the cannula moved freely about in what was naturally believed to be the trachea but proved to be the abscess cavity.[4]

[1] Verhandl. Phys. Medic. Gesellsch., Würzburg, 1868, i, 27.

[2] Casper's Wochen., 1842, p. 192. Also in Dittrich, Prag. Vierteljahr., 1850, iii, p. 129. [3] Bayle, Nouveau Journ., t. iv, p. 37.

[4] Green, Brit. Med. Jour., Dec. 17, 1870, p. 649, and Marsh, St. Barth. Hosp. Reports, iii, 368, report cases (not following fevers, however) in which the tube was inserted in the cellular tissue in front of the trachea.

In three cases of edema the cannula was removed in six, eight, and nine days respectively. But after such serious loss of substance and extensive organic mischief as are involved in the cases of perichondritis, it is not a matter of wonder that the stenosis of the larynx is generally permanent. In 17 cases of probable or actual perichondritis, the cannula was removed in one case after seven months, but in the other 16 cases, when last seen, the patients were still wearing them. Once, after seven years' use, a piece of the cannula wore away, broke off, and fell into the trachea, whence it was successfully removed by Albers. Busch, Russell, and others have attempted to dilate the stricture, both from above and below, but without any success.[1]

IV. Gangrene.

The cases of gangrene may be divided into two classes: A, those from pressure; and B, cases of spontaneous gangrene.

A. Those from *pressure*, or the ordinary *bedsores*, are not peculiar to the continued fevers, as is well known, but arise from any prolonged debilitating disease or accident. They are more common, therefore, in typhoid fever than in typhus, on account of its greater duration. But they not infrequently follow typhus, if it be prolonged by any complication. The only points in addition to the greater danger of pyemia, to which I desire to call attention, are as follows:

First, as pointed out by Nélaton,[2] Blandin,[3] and others, if they penetrate deeply they may destroy the ligaments uniting the sacrum and coccyx, and so penetrate into the spinal canal and set up meningitis, etc. This complication, I believe, arises much more frequently from bedsores, whatever their cause, than is generally known. Recently I have had two instances: one arising from a bedsore following confinement, the specimen which I show you, and which I owe to the courtesy of Dr. Schell, my colleague at St.

[1] By the courtesy of Drs. Otis and Woodward, of the Army Medical Museum, the casts and specimens of Dr. Buck's case, p. 342, and of several cases of laryngeal stenosis, were shown.

[2] Path. Chir., Paris, 1844, i. 256, 257.

[3] Anat. Top., 2me ed., p. 437. See also Charcot, Mal. du Syst. Nerveux, 2me éd., i, 89, 90.

Mary's Hospital; and the other in a boy who injured his knee and died some weeks after from tetanus induced probably by this complication. I have found six cases of tetanus recorded; four following typhoid and two typhus. Four of them were females. In one it was clearly caused by a bedsore.[1] Four of them died, but the two women who recovered had had menstrual irregularities, which probably caused the alleged tetanus.[2]

Secondly, large bedsores, as in two cases reported by Chenu in the Crimea,[3] may greatly hinder free motion of the legs and trunk by the extensive cicatrices.

Thirdly, the treatment first proposed, I believe, by Brown-Séquard, of ice poultices for fifteen minutes, followed by hot flaxseed poultices for two to three hours, often stimulates the most indolent bedsores to heal with surprising rapidity. During and since the Civil War I have repeatedly and successfully tried this plan of treatment.

Sometimes gangrene results from the slightest pressure, as in a case reported by Stokes,[4] in which there were 30 such spots, two or three new ones appearing every morning at points of such trifling pressure as where the mammæ leaned on the arms, or one leg on another, and a black hand appearing where the face had rested on the hand. Strange to say, the woman recovered after a month's abdominal decubitus. For such cases Liebermeister recommends an almost continuous and complete bath, the body resting and reclining on sponges.

B. But the cases of so-called *spontaneous gangrene*, though less frequent, are of far greater interest from a surgical point of view. They vary greatly in frequency. Thus, neither Flint nor Trousseau ever saw a case; Nélaton does not name fever as a cause of gangrene; Murchison, though he has seen a few, does not cite a single English post-mortem. Yet Estlander[5] reports 34 cases, and I have collected in all 113 cases. The frequency varies in proportion to

[1] Maclagan, Edinb. Med. Jour., 1867, 297.

[2] Delairière (Jour. de Méd. Chir. et Pharmac., xiii, 19) reports also a case of hydrophobia, which became ataxic, following a quotidian fever. The patient had been bitten by a healthy dog three months before. R. Reid (Pathol. and Treat. of Fever, Trans. Queen's Coll. Phys. Ireland, iii, 41) alludes to the similarity of hydrophobia and the excitable stage of fever.

[3] Rapport, pp. 520, 524.

[4] On Fever, Phila., 1876, p. 210.

[5] Archiv klin. Chir., 1870, 453.

the severity of the case and of the epidemic, and especially to the preceding conditions as to bodily nourishment, mental depression, and general mode of life. In former wars especially, from the time of Thucydides to that of Napoleon, fierce epidemics, especially of typhus, have deciminated armies and often displayed a most frightful tendency to gangrene. But of late, whether in civil or military practice, if we may judge from the scanty gleanings I have been able to obtain from the journals and the experience in our own Civil War as well as in the late European wars, the condition of the sick has been so ameliorated that gangrene is happily a rare complication.

The history of the extraordinary series of cases reported by Estlander well illustrates these predisposing causes. In Finland, a financial crisis and a series of bad harvests from 1862 were followed in 1865–67 by sporadic cases of fever, mainly typhoid; but from the thrifty habits of the people and governmental support, the epidemic was at first neither severe nor extensive. Then came the cold and rainy summer of 1867, followed by a very bad harvest. That winter typhus raged in almost every household, so that often the well were not numerous enough to nurse the sick. The death-rate rose from 2.74 per cent. to 7.69 per cent. Instead of an annual increase in the population of over 15,000, it decreased in 1868, nearly 94,000; and this in a population of less than 2,000,000. Of 105 doctors, 30 sickened and 8 died. Up to 1868 not a case of gangrene occurred, but in the first seven months of that year 28 cases occurred. Then came the bountiful harvest of 1868, and by August the epidemic had almost disappeared. Yet the lingering effects of the previous want were seen in six later cases of gangrene. But such an experience is altogether exceptional. From other writers I have rarely obtained more than two or three cases.

Estlander's 34 cases were all, except one, from typhus; but of the remaining 79 cases, 43 followed typhoid, 22 typhus. The influence of age is not very marked, as is seen in the fact that of 76 cases, 8 occurred before fifteen, 27 from fifteen to twenty-five, and 32 after twenty-five years of age. But sex, as usual, has a marked determining influence. Of 81 cases, 56 were males and 25 females. This is the more curious when we consider that the

number of deaths in men, and therefore presumably of cases, does not hold at all the same relation. In 1868, in Finland, 31,000 males and 28,000 females died; yet of 31 cases of gangrene of the legs, 25 were males, and only 6 females. The site of the gangrene is very suggestive also. In 5 cases it was in the ears, 10 in the nose, 27 in the face, neck, and trunk, 5 in the arms, 7 in the genitals, and 72 in the legs; that is, of 126 localities, in 77 it was in the extremities, and in 22 more in other peripheral districts of the vascular system—ears, nose, and genitals.

As far as the pathology of the cases is concerned, they may be divided into two classes: (1) Those with a discoverable clot and (2) those without such a clot. Murchison believes that all cases of spontaneous gangrene arise from arterial thrombosis, but the careful post-mortem examinations of Estlander and others show that, at least in the larger visible vessels, sometimes no such thrombus exists.

1. Those *with clot.* The cause of such clots, as Humphrey [1] and others have shown, is not the condition of the blood-vessels. But seldom have I found it stated that the arterial walls were diseased, and when they were it was presumably a secondary process, the result and not the cause of the clot. Few, if any, pathologists will now attribute such results, with Bourgeois,[2] to a metastasis, especially when arising in convalescence, as these so frequently do. Gigon [3] has attributed them to chemical alterations in the blood which give it an irritative character, and this, with friction at points of curvature, produces inflammation and coagulation. If so, a fair proportion of cases should be seen in the upper extremities, where the same irritating blood circulates and similar curves exist. How rare this is we have already seen.

Although the precise factors in determining the thrombosis in any individual case may be somewhat doubtful, as also why it is frequent in one epidemic and rare in another, apparently frequent in Germany and rare in Britain, and especially rare in the United States, yet three causes clearly exist which may vary *inter se* in producing the result: (1) The altered blood; (2) the weakened heart; and (3) the

[1] Brit. Med. Jour., 1859, p. 582.

[2] Arch. Gén., Aug., 1857, 149, and L'Union Méd., 1861, xii, 80, 240.

[3] L'Union Méd., 1861, xi, 577, 611, and xii, 127.

mechanical difficulties in carrying on the circulation, especially in distant parts.

That the blood is profoundly altered, and probably has an increased coagulability, is conceded. In some cases, even, air is found in the veins, as noted by Crisp[1] and Lebert.[2] That every such change, besides its depressing effect upon the nutrition, and therefore upon the vitality of the tissues themselves, would interfere more or less with its circulation, and consequently predispose to thrombosis, is most probable. But when we look at the seat of the cases of gangrene of both varieties which are under consideration, I think the conclusion is inevitable that the last two causes are the more immediately determining factors.

The heart, as Stokes showed, is softened in its texture, and therefore weak. Hayem [3] has shown that myocarditis is extremely frequent, and not rarely involves the endocardium. From the sixth to the fourteenth day is its weakest period, and not only is the general force of the circulation diminished at this time, but, all the blood not being squeezed out of its cavity, clots may form in the heart, and then, or at a later period, when the heart regains somewhat of its force, be washed into the circulation and lodge as emboli. Such seems to have been the origin of the clot in a remarkable case related by Patry.[4] A decolorized adherent embolus was found high up in the left external carotid, on which a secondary thrombus had formed nearly down to the bifurcation of the primitive carotid. Pain appeared from the jaw to the temple on the twentieth day of typhoid; two days later the ear was cold and violet, the artery pulseless, and the gangrene rapidly extended to the entire left side of the head and face, involving even the bones. I have found, however, but eight other cases in which the embolic nature of the primary obstruction was clear; but often the want of a minute examination of the clot renders the report useless, and sometimes, probably, the primary embolus is so overshadowed in size and importance by the secondary thrombus as to be overlooked.

[1] Dis. Blood-vessels, p. 18.

[2] Prag. Vierteljahr., 1858, i, 33. Moorehead, Trans. Med. and Phys. Soc., Bombay, 1843, p. 68, also reports a case.

[3] Leçons clin. sur les Manifest. Cardiaques de la Fièvre Typhoide, 1875, 49.

[4] Archiv. Gén., 1863, i, 144.

The third factor, the mechanical difficulties of the distant circulation, combines almost inextricably with the weakened heart in producing the spontaneous coagula or thrombi. It is, nevertheless, clearly the principal factor in precipitating the gangrene in the lower extremities. Not only, however, are the inferior parts of the body thus involved in gangrene, but the frequency of venous thrombi, and the resulting phlegmasiæ in the same region, is a strong argument in the same direction. Bouchut [1] found in 51 cases of non-puerperal venous coagula that 44 were situated in the pelvic, femoral, or tibial veins. I have memoranda of 63 cases of venous coagula following the continued fevers in which the site is stated. Only two cases involved the upper extremity alone, and both were followed by gangrene ; one involved both the arm and leg ; all the other 60 cases were limited to the lower extremities.[2] Both forms of coagula—the arterial and the venous—form most frequently during or just after the period of greatest cardiac weakness—a weakness felt most at such distant points, as the legs. Of 18 arterial cases 12, and of 43 venous cases 24, occurred in the second and third weeks of the fever. Moreover, the preceding circumstances—such as famine, individual poverty, and the deprivations of war—are such as impair the nutrition and the circulation in the peripheral districts of the body. The coagulation also takes place at points mechanically favorable to slowing of the currents—*e. g.*, the bifurcation of arteries and the valves in the veins.[3] In the veins, at least, as described by Humphrey, the clots are sometimes laminated, the outer layers of decolorized fibrin, and therefore the oldest, and the center, a bar-like recent coagulum of dark or black blood.

Once that the obstruction exists in the artery, it extends by additional coagulation, so that the collateral circulation may be widely and rapidly cut off. The progress of the clot can often be watched from day to day by the progressive annihilation of the pulse—first, for example, in the tibial, then in the popliteal, then in the femoral or higher—

[1] Gaz. Méd. de Paris, 1845, p. 241.

[2] See a very interesting case which got well after a second attack of typhus thirty years later, Stokes on Fevers, Phila., 1876, p. 249 (republished in Med. News and Library).

[3] See a carefully reported case of Phlegmasia by Cole, Med. Times and Gaz., 1875, i, 5.

and by the parallel progress of the gangrene. In cases of recovery this cessation of the pulsation and the hard tender cord in the course of the vessels are, of course, the only, but sufficient, proof of their occlusion. That gangrene follows so much more frequently in febrile thrombosis than after the traumatic thrombus which accompanies ligation is not surprising, in view of the condition of the blood, the general enfeeblement, and the more wide-spread arrest of the collateral circulation. Yet, on the other hand, pyemia, which has so much to favor it, especially in the cases of venous thrombi, is a rare sequel. Even when it does follow, it is in most cases apparently the secondary result from the septic influences arising from the gangrenous parts.

The circulation in the artery being cut off, it is not strange that clots should follow in the veins; but even where both are obstructed moist gangrene rarely follows. The foot especially generally mummifies. But this is not always the case. Occasionally the gangrene is moist from the beginning, from early obliteration of the vein, or, having begun as dry gangrene,—*e. g.*, of the foot from a popliteal clot,—suddenly both the femoral artery and vein may be obstructed, and a moist gangrene of leg or thigh be added. Gangrene from venous obstruction alone is very rare. It is more apt to follow in the arm than in the leg.[1]

Coagulation of the blood may also be caused occasionally by direct mechanical causes in fevers, as in a case given by Jaesche, in which a swollen gland surrounded the common iliac artery at its bifurcation and caused a clot, probably by direct pressure or by induced arteritis.

2. The second variety of spontaneous gangrene is that in which *no clot* apparently exists—certainly no such clot as is commonly designated either an embolus or a thrombus; that is, a local clot of some size in an arterial trunk, which cuts off the circulation in the tissues supplied by its branches. But even in these cases I believe that the conditions affecting the circulation, already so fully considered, will more readily and rationally explain the causation of the gangrene than any specific action of the indefinite though

[1] Hueter (Virchow's Archiv, xvii, 48) records, however, a very interesting case of gangrene of the right leg following a spontaneous clot without assignable cause, the vein wall being healthy.

undoubted poison of the fever. Coagulation I believe still to be the cause, but not in the larger trunks. It begins, rather, as a blood stasis in the capillary circulation. The parts in which the often extensive coagulation takes place are at once struck with gangrene, and, as the blockaded vessels themselves are all involved in the general destruction of the gangrenous tissues, all evidence of the nature of the lesion is thus destroyed. This form of gangrene occurs generally in the nose, ears, penis, perineum, labia, feet, and occasionally the fingers; that is, in parts supplied by no one large vascular trunk, but by many smaller branches—not only in parts distant from the center, such as the feet, but in parts which lose their heat most readily by reason of their thinness and small size, and parts irritated, it may be, by local discharges. Very probably, also, it may be due to the fatty degeneration of the smaller arteries, as observed by Hoffmann, Zenker, and Ponfick. Raynaud[1] (and, following him, Fischer and Estlander) ascribes it to a spastic ischemia, from contraction of the arterioles. The frequent bilateral or symmetrical character of this variety, to which Raynaud has called especial attention, would indicate at least the probability of some such central cause. Whether, if it exist, it be the direct result of irritation of the nerve-centers, as is seen in the other nervous phenomena of fever, or whether it be a reflex spasm caused by the circulation of deteriorated blood, similar to that to which Dr. George Johnson has attached so much importance in Bright's disease, we can at present only surmise.

The symptoms of gangrene are marked and characteristic. Toward the end of the fever, especially in the third week, or early in convalescence, as weakness is giving place to strength, and the brightest hopes of speedy recovery are cherished, sudden, severe, and persistent pain is felt. Usually it is at the seat of the impending gangrene, though not uncommonly at the clot itself, radiating thence to the periphery. In the lower extremities it is often felt in the ball of the great toe or in the heel. It is followed by numbness, coldness, loss of sensation, and sometimes of motion, and in a short time discoloration and all the other usual evidences of gangrene appear. Sometimes, but not

[1] De l'Asphyxie locale et de la Gangrène Symmétrique des Extrem., Paris, 1862.

usually, these local symptoms precede the pain. If the distal vessels be examined, the pulsation will be found feeble or utterly extinguished, while higher up, at the seat of the obstruction, they will be changed into moderately firm but tender cords, in which we may sometimes differentiate the artery from the vein—an important point in prognosis. Week by week, sometimes day by day, the process of the coagulum may be traced upward by the abolition of the pulsation and by the upward march of the gangrene. If old cicatrices exist, they will be among the earliest parts to yield. Blebs may form in the early stages, but most frequently they will dry up, and the parts will mummify, although, as already indicated, moist gangrene may supervene if a large clot form higher up, or if the veins are extensively obliterated, thus involving great masses of moist tissue, such as the thigh, in sudden ruin.

Life may be rapidly destroyed, as, in a case recorded by Barker and Cheyne,[1] in two and one-half hours after gangrene began in the nose; but more commonly days or weeks will elapse, during which nature, as usual, makes a powerful effort to rid herself of the dead parts by the establishment of a line of demarcation. On the establishment of this the pain often ceases.

If recovery follow, the circulation is carried on by collateral branches, or, in very rare cases, the artery again becomes partially pervious.[2] This last result Humphrey has shown to be not infrequent in veins, and Pètres[3] has recently elucidated its mechanism through the extension and coalescence of the vasa vasorum.

In the variety of gangrene without a thrombus, the symptoms will vary somewhat. It is not so uniformly in the lower extremity, and is much more frequently symmetrical. If small in extent, pain is not apt to be a leading feature. The onset is often earlier, and from the nature of the case its progress is sharper, and its limits much more quickly defined, so that usually, within a few days, at least, the boundary of the gangrene is pronounced, since it does not progress with any gradually growing thrombus. For the same reason it less frequently returns in the stump after

[1] Account of the Fever lately Epidemic in Ireland, London, 1821, vol. i, p. 232. [2] Patry, Archiv. Gén., 1863, i, 136.

[3] Edinb. Jour., Aug., 1875, p. 175, from Le Progrès Méd.

an amputation. Its area, also, is usually much less than in those cases in which a thrombus exists, rarely extending in the leg beyond the foot or ankle; and it rarely involves surrounding parts to a large extent if it occur in the nose, ear, genitals, etc. Sometimes, however, it may extend more widely, as in a case of typhus and starvation mentioned by Lyons,[1] in which the patient walked to the workhouse, and on baring his chest the whole of the right side was "a dark, olive-green, jelly-like, tremulous mass." The abdominal wall is sometimes similarly involved. The probably irregular area in which the stasis of the blood will take place in this form also accounts for the great irregularity generally seen in the line of demarcation; whereas, if a clot exists, it is apt to be fairly even. This sudden history is usually followed by a speedily decided issue. Death follows quickly, or reaction and recovery set in within a short time, instead of hanging in the balance for months.

The *results* of spontaneous gangrene vary much, according to its situation and extent. In the extremities, if life be saved, the result is usually an amputation, either by nature or by the surgeon. In the nose, it may perforate the septum or destroy the entire organ to a greater or less extent. During some civil, as well as military, epidemics of typhus, this seems to have been a favorite spot for its beginning, so that the disease was popularly known early in this century as the "Blue Nose,"[2] and inspired terror whenever it appeared. In 1834, Mauthner says, it was an extremely common result, seen in all the military hospitals, and "all hope was gone as soon as this dreadful symptom was seen." Another not infrequent form is noma, or cancrum oris. This is especially frequent in children and in the army. Murchison speaks of it in the Crimea as frequent and invariably fatal; Chenu, however, in his report does not name it. Its ravages are extremely extensive, often involving even the bones.

The ear, also, and the eyelids are sometimes destroyed.

[1] On Fever, p. 191.

[2] See Mauthner, Ueber das typhöse Fieber mit Nasenbrand, Hufeland's Jour., 1834, lxxviii, 46; Kraft Ueber Typhus Bellicus u. die blaue Nase, Hufeland's Jour., 1815, xli, 81; Gutberlet, Die blaue Nase bei dem Typhus Bellicus, Hufeland's Jour., 1816, xlii, vi, 101; Wendelstädt, Die blaue Nase beim Typhus Bellicus, Hufeland's Jour., 1816, xliii, v, 131, and Barker and Cheyne, *loc. cit.*, i, 232.

From each of these, singly, or all together, the most frightful deformities often follow, which require the utmost ingenuity in the plastic operations necessary to remedy them. In many cases the gangrene is local and subcutaneous, producing necrobiotic masses of tissue, which are, I believe, often, if not generally, the cause of the abscesses so commonly seen in all parts of the body. Sometimes even the mediastina are opened,—the anterior from the chest wall, the posterior from the deep tissues of the neck[1],—unless, by a timely surgical operation, the danger be averted.

The male genitals are occasionally destroyed to a greater or less extent. Except the organic destruction, no special result follows, except, possibly, hemorrhage, for one case is recorded of death from a hemorrhage of ℥xxx from the scrotum.[2]

That the perineum and the female genitals are not more frequently the seat of gangrene is rather surprising, when we consider the neglected condition of many of the patients and the constant soiling of the parts, as a result of unconscious and unavoidable discharges, especially in females. The troubles of the female generative organs are either distinct external gangrene, or gangrenous ulcers in the vagina. I have found 9 cases,—8 from typhoid and one from typhus,—all in young persons from seventeen to twenty-seven years of age, except one of thirty-four. In 6 of the cases there was gangrene of the labia, extending sometimes to the perineum and the thigh. At least one case was followed by contraction of the vulva.[3] In another, reported by Guéneau de Mussey,[4] there was complete occlusion of the vagina and menstrual retention, necessitating puncture, with a fatal result. The ulcers are generally on the posterior wall of the vagina, and in three cases recto-vaginal fistulæ have resulted. One is reported by Lebert[5] in which, when convalescent in the seventh week, chill, fever, and diarrhea set in, and four weeks later the fistula was discovered by injection. It was situated in front of the hymen, and was as large as a five-centime piece. A

[1] See Fraentzel, Berlin. klin. Wochen., 1874, xi, 97; Werner, Med. Corresp. Württemb. Aerztl. Verein, Stuttgart, 1859, xxix, 76, and Hoffmann, *loc. cit.*, p. 388.

[2] Murchison, p. 194.

[3] Russell, Glasgow Med. Jour., 1864–65, xii, 165.

[4] Gaz. Hebdom., 1867, 652.

[5] Anat. Path., ii, 307, and pl. cxv.

month later she died of pelvic peritonitis. A second is reported by Liebermeister.[1] It was caused by the sloughing of a large piece of the recto-vaginal septum, in mass. The large fistula thus produced healed without operation. The third case has been under my own observation in St. Mary's Hospital for three years past, and is the only case I have found of both recto-vaginal and vesico-vaginal fistulæ. [For this case in full, see Case II, p. 80.]

The perineum suffers mostly in males as 8 to 3, while in 2 cases the sex is not stated. Typhoid was the cause in 11, typhus in 2. Although not all cases of gangrene, they may be surgically grouped together, since all but one produced perineal fistulæ. The exception[2] was a case fatal from a large abscess around the membranous urethra. Three fistulæ were caused by necrosis of the pelvic bones or sacrum, and nine by gangrenous ulcers, which sloughed not only externally, but in five certainly communicated with the rectum, and probably did so in others. Except two cases of twenty-one and twenty-two years of age, they all occurred (when the age is stated) from thirty-nine to seventy-four years of age, later in life than most of the other sequels. They arose from the third to the seventh week,—that is, during distinct convalescence,—and to this is probably due the fact that 10 recovered and 2 died, one from the peri-urethral abscess, the other from hemorrhage upon sloughing into the rectum.

The question of *treatment* of gangrene is, after all, the most important in a practical point of view, and is divided naturally into the preventive and remedial. The general supporting treatment of the disease is, of course, the most important preventive. Next, a careful and repeated examination of the body, especially the parts most likely to be attacked. If gangrene is specifically threatened, stimulation of the circulation, both at the center and at the threatened spot, is imperative. To stimulate the center, alcohol in liberal doses is the best remedy, and two extremely instructive cases are given by Stokes.[3] In one, "the surface was cold, and the pulse imperceptible. From the middle of the calf of each leg downward over both feet the sur-

[1] Ziemssen's Cyc., Amer. ed., vol. i, p. 184.
[2] De Change, Arch. Belg. de Méd. Mil., 1861, xxviii, 126.
[3] On Fever, Phila., 1876, p. 205.

face was black, the skin hanging in loose wrinkles, giving an appearance as if the patient had on a pair of black socks." Sixteen ounces of brandy in the first eight hours saved his life. Digitalis might also possibly be used with advantage. The peripheral circulation must be stimulated by such means as will assist the threatened circulation by inducing alternate dilatation and contraction of the arterioles. Permanently wrapping up the part in cotton, and other similar means, will but assist permanent vascular dilatation and stasis. The alternation of the two is the condition of health, and its artificial production will tend to restore healthful reaction.

Chapman's ice- and hot-water bags to the spine, alternate heat and cold directly to the parts, with proper friction and stimulating liniments, at once commend themselves to us. The constant current battery also may prove an extremely useful aid, since it dilates the deep as well as the superficial vessels, and will aid the collateral circulation.

But suppose gangrene actually occurs, what then? Estlander gives most judicious counsel here. W emust remember that good results follow both to life and limb *without* operation, especially if the gangrene be limited and the patient not too exhausted. We must not, therefore, be rash in our interference. If amputation has to be done, the question as to where it should be done depends on the probable extent of the gangrene; as to when, on the line of demarcation. In the non-thrombotic cases, as the line of demarcation is usually established within two or three weeks, and the disease is not then likely to be progressive, the amputation may be done but little above or even through it. It is, therefore, usually best to wait for its formation. In the thrombotic cases, the clot and the gangrene *are* apt to be progressive. Until the line of demarcation forms, therefore, it is impossible to say precisely where the disease will stop. Yet we can gain some idea of the probabilities of the case from past experience.

If the clot extend no further than the popliteal, the limb may escape gangrene altogether, and if it follow, I have found it limited in 9 cases—to the foot 4 times and to the upper calf in 5; if the clot extend into the femoral, I find the gangrene extended to the upper calf in 6 and to the thigh in 4; if the clot extend above Poupart's ligament, I

find in 10 cases it was limited to the foot in 1, the calf in 3, and extended above the knee in 6. The results of amputation are good, giving 21 recoveries to 21 deaths, but the recoveries are largely after amputations in the foot. Before demarcation was established, 5 out of 8 died; after demarcation, 12 out of 22: a mortality, respectively, of 63 and 55 per cent. As a rule, therefore, wait for the line of demarcation, but amputate soon after its appearance; but if danger of septic poisoning or of speedy exhaustion should appear, amputate at once, at or above the probable limitation of the disease, which, if the femoral be free, will not be, in the majority of cases, above the tubercle of the tibia; but if the femoral be involved, amputation would probably be more dangerous than the expectant treatment. As dead parts slough, they should be removed to prevent septic poisoning. Fortunately, there is but little danger of hemorrhage, either primary or secondary, in the thrombotic cases, since the arteries are all plugged securely.

Of course, the ordinary treatment of the gangrenous ulcers and abscesses, especially of the perineum and genitals, should be pursued, but I would particularly urge the importance of free incision, especially in abscesses in the vicinity of the anus, and the use of detergent and stimulating washes in the vagina in case of sloughing of the labia, in order to prevent in both cases the establishment of fistulæ.

V. Hematomata.

The muscular system suffers, in typhus and typhoid fevers, in common with almost every other tissue of the body, undergoing a peculiar form of degeneration, resulting sometimes in rupture and the formation of hematomata. These, although not so strictly surgical as some of the other diseases noted, yet, as their proper surgical treatment is so important, I shall notice briefly.

Apparently, the first published case was observed by that shrewd surgeon Velpeau,[1] in 1819, in the post-mortem examination of a soldier at Tunis. "Rupture of the muscles of the belly," says he, "is not surprising. The organs become so fragile in advanced stages of putrid fever that their rupture is a phenomenon which is easily conceived

[1] Dict. en trente Vol. Art. l'Abdomen, Rupture de.

when the patient in his delirium moves so irregularly." In 1844 Rokitansky noted their relation to typhoid. Virchow studied them also in 1857.[1] In 1864, however, Zenker[2] first studied the subject thoroughly. Since then, but especially within the last five years, they have been frequently observed or studied, mostly, however, from a pathological standpoint.

The muscular changes to be described are so frequent as to be almost an essential part, at least, of typhoid. Yet they are not peculiar to these fevers. They are said to have been met with in phthisis, scurvy, scarlet fever, cholera, pneumonia, dysentery, measles, tetanus, Bright's disease, cerebro-spinal meningitis, muscular traumatism, and I have seen a similar change in the muscles of the abdomen in cases of large ovarian tumors.

There are two independent forms: (1) A granular degeneration of the muscular fibers, which is least frequent; and (2) a waxy change, which is by far the commonest. Under the microscope the muscular tissue presents a glassy, translucent, slightly opalescent, shiny appearance, the fibers being swollen to even double their usual size and changed to fragile cylinders. Sometimes the muscular tissue resembles even the flesh of fish. The nature of the change is as yet greatly disputed. Erb,[3] Bernheim,[4] and others attribute it simply to post-mortem imbibition; Hayem,[5] to proliferation of the tunica intima, which, with granulo-fatty change in the arterial walls, produces an obstructive arteritis; Zenker ascribes the degeneration to the disturbance of a center which regulates the nutrition of the muscles; Waldeyer,[6] Hoffman,[7] Ranvier, and Weihl[8] believe that it is a coagulation of the myosin; and Liebermeister that it is due to the long-continued high temperature. Whatever the cause, the muscles become extremely fragile, and when they are called into play by the distention from meteorism, by

[1] Wurzburg, Verhandl., vii, and Virchow's Archiv, iv.

[2] Ueber die Veränd. der willkürl. Muskeln im Typhus Abdom., Leipzig, 1864.

[3] Virchow's Archiv, 1868, xliii, 108.

[4] Gaz. Méd. de Strasb., 1870, No. 7.

[5] Gaz. Méd. de Paris, 1866, 698.

[6] Centralbl. med. Wissensch., 1865, 97, and Virchow's Archiv, 1865, xxxiv, 470.

[7] Virchow's Archiv, 1867, xl, 505.

[8] Virchow's Archiv, 1874, lxi, 253.

the efforts at coughing and other violent respiratory acts, by defecation, rising in bed, the movements of the legs, etc., they rupture with the greatest ease. Spasm or direct violence does not seem to have been noticed in any case. The arteries, which have also undergone an analogous change, are involved in this rupture, and muscular hemorrhages result. These assume three forms, according to their size and mechanical limitation: (1) Ecchymoses; (2) diffuse infiltration into the muscular tissue, soaking it with blood; or (3) distinct hematomata, the last being the most important and probably the most frequent. The effused clot, at first hard, well defined, and sharply limited, gradually softens, and not infrequently suppurates, thus producing serious abscesses which, unless opened, may even burst into the peritoneal cavity.[1] Meanwhile the swollen muscular fibers gradually undergo reabsorption, until finally they disappear entirely, and a new formation of cells takes place in the perimysium, which, according to Hoffmann, first become spindle-shaped, then coalesce endwise with one another, and gradually assume the appearance of striated muscular fiber. Complete repair is then effected. The resemblance of these spindle-shaped cells, which are nascent muscular fibers, to the muscular fiber-cells is most striking, and seems to form a link connecting the two forms of muscular tissue, the striated and non-striated, such as I have long taught to be probable.

Almost all of the muscles may be thus invaded, but the favorite seats both for the degeneration and the hematomata are in the recti abdominis and the adductors of the thigh, then in the pectorals, and, as Hoffmann has noticed in 16 cases out of 22, in the diaphragm. The influence of the phrenic lesion in enfeebling the respiration is, perhaps, more serious than has been recognized. Zenker gives the adductors the first place, and Hoffmann reports the adductors involved in the degenerative changes in 75 out of 107, the recti in 87 out of 127. While this may be true of the degenerative process, yet the hematomata are certainly most frequent in the recti. Of 60 positions in cases I have collected from every side, they were in the recti in 27, and

[1] Wenzel Gruber, in Jacops, Etude clinique sur les Abscès. musc. qui surviennent pendant la Convalesc. de la Fièvre Typhoide, Thèse de Paris, 1873, p. 42.

the "abdominal muscles" in 9, in the adductors but 5 times, and the upper extremities but twice.[1] If in the adductors, they may burrow so as even to strip off the periosteum from the bone. Hematomata are even found n the interventricular septum of the heart itself.

Stokes suggests that febrile deafness and hoarseness may result from a similar degeneration of the muscles of the ear and the larynx. There are no post-mortem examinations on which to found such a hypothesis, and the fact that hoarseness and deafness are so often not seen, and that, as I have shown,[2] other and sufficient causes are found, at least in the larynx, render the idea scarcely tenable.

Typhoid was the preceding fever in 44 out of 46 cases, but the severity of the fever seems to have but little influence. Nineteen out of 25 cases occurred from fifteen to twenty-five years of age, and 22 were males as against 8 females. They rarely appear before the third week, since the muscular fragility is then at its height. Of 23 cases I find 19 arose in the third, fourth, and fifth weeks of the fever. Regeneration of the muscles usually begins in the third or fourth week, and is accomplished by the seventh, after which time they do not appear. Their period of development is, therefore, quite sharply defined by the anatomical history.

As in dislocation of the hip-joint, the symptoms are often nil. Indeed, of 47 cases, I find 10 were wholly unsuspected until revealed at the post-mortem. The position of the tumor accounts in part for this. Both in the thigh and in the abdominal wall they are almost always in the posterior part of the muscle—a position due, probably, to the effect of gravity in the recumbent posture. Often, indeed, they are so deep as to extend to the pelvic and iliac muscles, and, in two cases, under the serous coat of the bladder. Hence there is usually little or no discoloration of the skin, though Foucault [3] reports a case with ecchymosis in the hypogastrium, extending later to the scrotum, thighs, and buttocks. Swelling is only reported 13 times, and fluctuation but 10 times. Suppuration and softening are but rarely attended with any special fever. Pain is

[1] In the recti they are, I believe, invariably below the navel, possibly on account of the absence of the support derived from the linæ transversæ.

[2] *Ante*, p. 353.

[3] Bull. Soc. Anat., 1869, 498.

mentioned in 14 cases. Flexion of the legs to relax the abdominal wall, which we would suppose to be frequent, is named but once. The size of these blood tumors varies from that of a bean to that of an orange. If small, they may be obscured by meteorism; the symptoms then being so indecisive, as in many of these surgical sequels, the necessity for frequent and rigid physical examinations is at once apparent. If a sudden and fixed pain exist in the recti below the navel, or even if movements be only hindered or uneasy and painful, a close examination should be made, and if a tumor or only hardness be found, it should be carefully scrutinized from day to day, especially for the pasty feel and other signs of edema and of fluctuation. The differential diagnosis is not usually very difficult. The most likely error, if in the rectus, is that of mistaking it for a distended bladder, but the catheter will at once unmask this error. If in the right iliac region, it may be mistaken for perityphlitis; or in the adductors, for a simple abscess; but as in point of practice the treatment of all three would be more or less similar, the error is of less moment than might be supposed. From aneurysm, an abdominal tumor, and peritonitis, the differential diagnosis is sufficiently easy.

The *treatment* is important, especially when we consider the results. Of 13 cases opened by incision, only 2 died; of 34 in which there is no mention of an operation, all died. If small, they will either be overlooked until the post-mortem reveals their existence, or, if recovery takes place, absorption of the clot and regeneration of the muscle will follow, independent of treatment. If large, every possible effort should be made by poultices, etc., to bring about early softening, and as soon as softened they should be opened. That the aspirator may be of service is probable, but as yet it is, I believe, untried.

VI. Parotitis.

Parotitis is occasionally an exceedingly important surgical complication, whose onset is always to be dreaded, lest it bring in other evils worse than itself. Murchison believes with Graves that the inflammation begins in the areolar tissue between the lobules of the gland itself, but Hoffmann

has unquestionably shown that, at least in typhoid, the pancreas and all the salivary glands are in a state of rapid cell-proliferation in nearly every case, and that parotitis proper is merely "an exaggeration of the changes that usually take place in this gland during typhoid fever, and bears the same relation to these changes that ulceration and perforation of the intestine do to the infiltration of the intestinal follicles." This exaggeration he believes to be due to the dense parotid fascia which compresses the gland. But this is not the only rôle this dense investing fascia plays. The compression of the swollen tissues not rarely produces gangrene, so that the entire gland may slough out in great masses like tow. In a case related to me by Dr. Grove, it involved both glands and proceeded so far that the fingers could almost meet behind the pharynx. The compression also is very favorable to thrombosis, which may extend to the brain by the diploic veins or even to the internal jugular itself. Necrosis and septicemia not rarely follow in its track. In two cases I have found facial palsy, from involvement of the seventh nerve. Facial deformity and ankylosis of the jaw are sometimes seen. In none does hemorrhage from the carotid appear to have followed.

The death-rate is largely increased in such cases, since of 352 cases, 125 died and 227 recovered; a mortality of nearly one-third. The sex is named in only 19 cases, of which 14 were males. Contrary to the fact in other complications, except in perineal fistulæ, this disease is most common after thirty. Of 211 cases, the average age, according to Murchison, was thirty-one and a half. It is certainly very rare in children, for I have found but 2 cases under fifteen. Typhus was the preceding fever in 352 cases, and typhoid in only 26. Most cases do not go on to suppuration, for of 101, I find 40 suppurated and 64 did not. The abscesses generally discharge by one or often by several openings, the external meatus being frequently one of them. As Nélaton has pointed out, even where it has thus opened, if we would avoid burrowing and other subsequent troubles, we must open it still more freely, in order to divide the parotid fascia.

Conclusions.

If now, by way of review, we cast our eyes back over the general results of all the complications and sequels we have studied, we may arrive at some useful and important conclusions.[1]

1. Typhoid, probably from its usually longer duration, is by far the more prolific source of such surgical troubles except parotitis, especially when we consider that many cases tabulated as typhus are really typhoid. Of 433 cases, typhoid was the preceding fever in 252, typhus in 119, and other forms of continued fever in 62.

2. The surgical troubles to be apprehended in typhus are mainly restricted to gangrene and laryngeal stenosis, 103 out of the 119 cases being due to these two classes of disease, while typhoid bears in its train any and all of the forms of disease described.

3. The age is about the usual age of greatest frequence of these fevers.[2] From fifteen to twenty-five years is by far the most frequent decade, counting 133 cases against 129 at all other periods of life. One singular exception is to be made—viz., the articular troubles, and especially dislocation of the hip, 21 out of 23 cases being under twenty years of age, of which 15 were in children under fifteen, in striking analogy to the frequency of coxalgia in children.

4. Sex is an unexpected and important factor in the predisposition to febrile surgical troubles. Of 303 cases in which the sex is named, 218 are males and 85 are females, or over 2½ to 1. What is the normal proportion of the sexes in fever it is difficult to determine. In nearly 6000 cases of typhoid, Murchison gives the proportions as precisely equal, and in over 18,000 cases of typhus the females were in a decided majority (8871–9267). Estlander's figures would give us a slight preponderance of males, while Liebermeister, in over 2000 cases of typhoid, gives 1300 males and 750 females.

[1] In this summary I have not included the cases of parotitis in the figures.

[2] Liebermeister gives the ages in typhoid as follows: fifteen to thirty, 1310; thirty to seventy-one, 394; total, 1704. None under fifteen were admitted. In typhus, fifteen to thirty, 39 per cent.

Murchison gives in typhoid: fifteen to twenty-five, 2752; all other ages, 3159; total, 5911; and in typhus, fifteen to twenty-five, 5332; all other ages, 12,806; total, 18,138.

Unfortunately, I omitted to tabulate the number of cases arising in military practice, which I am sure is not inconsiderable; but while this will account to some extent for the predominance of males, it could not be adduced in the cases of arthritis and dislocation, since most of the patients were children, yet the males were in the preponderance.

5. The period of development is not the initial period of the fever, but first, from its height to its close—that is, the complications, especially gangrene and stenosis of the larynx; and, secondly and most frequently, during convalescence—that is, the sequels. Of 240 cases, only 12 arose in the first week, 38 in the second, and 48 in the third, a total of 98. If we may assume that convalescence, on the average, begins at the end of the third week, then 142 occurred during convalescence, when health is apparently in the near future.

6. The lower half of the body is the especial seat of such surgical troubles. With the exception, of course, of the laryngeal cases and parotitis, of 307 cases 216 occurred in the pelvic region and legs, as against 91 in all other parts of the body. Moreover, the diseases attacking the upper half of the body are limited almost entirely to local gangrenes and caries and necrosis, and they are usually far less severe in type and more limited in extent than those in the lower half. Here whole limbs are blighted by gangrene; here occur most of the dislocations, the hematomata, the fistulæ; here the severest necroses and largest abscesses, and were we to add the long catalogue of bedsores and phlegmasiæ, the preponderance of the lower half of the body in importance would be still further increased.

7. The *diagnosis* is, in general, moderately easy. The danger is not that difficulty of diagnosis may obscure the case, but that the diseases may be entirely overlooked. They occur most frequently in parts of the body covered by the bedclothes; parts which require time and trouble to expose and examine in the routine of an ordinary visit. Moreover, the patient is frequently so apathetic and insensible to pain that he does not complain, or, if he do so, it is ascribed to the ordinary pains so frequent in the belly and legs in such fevers, or else to delirium itself.

Hence the most important hint I can give in the diagnosis—and where, indeed, does the same rule not hold good?

—is that time and trouble *must* be taken, and that no patient suffering from a continued fever, and especially from typhoid, should escape frequent, minute, complete physical examinations, in which every part of the body from head to foot should be questioned. Especially should the physical condition of the larynx, the belly, the legs, and the toes, and in children the hip-joint, be exactly ascertained. This should be done at least every second day, and that, too, not only in severe, but in mild cases, and not only during the fever, but especially in early convalescence, for it is in just such mild and convalescent cases that the wariness of the doctor is the patient's surest reliance. Particularly should attention be paid to hoarseness, or even to the slightest change in the voice, and the larynx be examined at once with the greatest care, from day to day, by the eye, the finger, and the laryngoscope, lest sudden edema or the more insidious and more fatal necrosis of the cartilages be impending. The eye should seize upon any hindered movements, even without discomfort, and no complaint of pain should fall upon a deaf ear, especially if it be in the throat, the belly wall, the buttock, the hip-joint, the legs, or the toes. True, it may mean nothing. It may be the vagary of a wandering mind. But it may also be, as we have seen, the herald of the gravest dangers, whose attack may be entirely repelled or their force broken by heeding this timely warning.

8. The prognosis is naturally unfavorable, yet not to the extent we would suppose from the addition or sequence of such serious diseases. Of 383 cases in which the result is named, 220 died and 163 recovered, a mortality of 57½ per cent.

9. Still more clearly, I think, after such a review, do we see the powerful influence of mechanical causes as the proximate factors in the production of such troubles, working in conjunction with the profoundly vitiated blood. With the exception, perhaps, of the almost constant muscular degeneration, and its not infrequent subsequent hematomata, these surgical results are not usually primary but secondary processes; not dependent directly on the fever poison, but its indirect and often distant results; not constantly seen, but incidental, indeed, often rare; not parts of the fever, but its complications and sequels.

Pathologically, all these results may be grouped into two categories—viz.: (1) Those in which a clot exists; (2) those without any clot.

1. Those in which emboli of cardiac origin or, more frequently, local thrombi exist are, unquestionably, most of the cases of extensive gangrene and phlegmasia. In many other cases in which such a clot is at present unsuspected, I believe that more careful examination will reveal its presence in the smaller vessels, and prove that, if venous, it may be a cause of edema glottidis, and, if arterial, of the local necrobiotic processes which result in necrosis of the bones, and probably of the cartilages of the larynx, and gangrene of the soft parts, with its abscesses, fistulæ, etc.

2. Those in which no clot exists, and yet edema glottidis, dropsy, and dislocation of the hip, gangrene, ulcers, necroses, perichondritis, and other similar troubles occur. These are especially often ascribed to the fever poison itself, acting locally, and producing, for instance, the so-called laryngotyphus, the abscesses and ulcers in the skin and subcutaneous tissues, etc., which are regarded as specific. While not denying this view outright, and especially in some cases, I feel still more strongly disposed to look upon them as allied disorders, the immediate results, as in the case of the pneumonia of fevers, of mechanical conditions, which produce a local stasis of the blood, followed by edema, low forms of inflammation, or gangrene. True, these results of fever are most frequent in severe cases and severe epidemics, in which the poison would be the most virulent, but it must also be remembered that such epidemics and such cases are themselves, as a rule, the result of exceptionally depressing pre-existing causes, such as famine and war, want and sorrow. Even simple inanition alone will produce identical results in many cases.

But it is especially when we consider the position of the troubles that this mechanical factor is apparent. Their posterior position, as is seen in the laryngeal ulcers, the perichondritis, the vaginal ulcers, the fistulæ, and in the hematomata the posterior surface of the recti and adductors, is most significant. Likewise is the fact that all such complications, as we have seen, are especially frequent in the lower extremities; that is, in parts mechanically un-

favorable to a ready return of the blood, and eminently favorable, if not to thrombosis, at least to stasis.

10. The *treatment* must be bold, but not rash; conservative, but not timid.

NOTE.—After the portion on Diseases of the Joints was stereotyped I received a letter from Dr. V. P. Gibney, of the Hospital for Ruptured and Crippled, New York City, giving the results in 860 cases of disease of the joints. The following is the only case which followed any continued fever, and it is not tabulated with the others:

"William H——, age twelve, presented himself at the outdoor department of the Hospital for Ruptured and Crippled, May 2, 1876. His general condition was good. The right hip was ankylosed, with the thigh abducted, semiflexed, and rotated inward, the trochanter carried upward, and he pelvis tilted to the right side. There was apparent shortening of the limb, but the real shortening was not ascertained. The thigh was atrophied three inches. Immense cicatrices of bedsores were found, one over each posterior superior spine of the ilium, one over the right natis, and one over each trochanter major, that over the right being the deeper, and covered by a scab 1¾ by 1½ inches.

"Prior to October, 1875, he was in perfect health, but was taken that month with typhoid fever, and lay very ill for six weeks, during which illness the bedsores formed, and during convalescence the deformity at the hip was observed. This history I obtained from the mother, who was very intelligent. At the time I saw him the disease was practically arrested."

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"It is arranged in such a manner as to be easy of access and comprehension. To any contemplating the study of histology and organography we would commend this work."

Fred's Soil Bacteriology

Published October, 1916

Soil Bacteriology. By E. B. FRED, PH. D., Associate Professor of Agricultural Bacteriology, College of Agriculture, University of Wisconsin. 12mo of 170 pages, illustrated. Cloth, $1.25 net.

McFarland's Pathology

A Text-Book of Pathology. By JOSEPH MCFARLAND, M. D., Professor of Pathology and Bacteriology in the Medico-Chirurgical College of Philadelphia. Octavo of 856 pages, with 437 illustrations, many in colors. Cloth, $5.00 net; Half Morocco, $6.50 net.

SECOND EDITION—published March, 1910

You cannot successfully treat disease unless you have a practical, *clinical* knowledge of the pathologic changes produced by disease. For this purpose Dr. McFarland's work is well fitted. It was written with just such an end in view—to furnish a ready means of acquiring a thorough training in the subject, a training such as would be of daily help in your practice. For this edition every page has been gone over most carefully, correcting, omitting the obsolete, and adding the new. Some sections have been entirely rewritten. You will find it a book well worth consulting, for it is the work of an authority.

St. Paul Medical Journal

"It is safe to say that there are few who are better qualified to give a résumé of the modern views on this subject than McFarland. The subject-matter is thoroughly up to date."

Boston Medical and Surgical Journal

"It contains a great mass of well-classified facts. One of the best sections is that on the special pathology of the blood."

McFarland's
Biology: Medical and General

Biology: Medical and General.—By JOSEPH MCFARLAND, M. D., Professor of Pathology and Bacteriology in the Medico-Chirurgical College of Phila. 12mo, 457 pages, 160 illustrations. Cloth, $1.75 net.

NEW (3d) EDITION—published January, 1917

This work is both a *general* and *medical* biology. The former because it discusses the peculiar nature and reactions of living substance generally; the latter because particular emphasis is laid on those subjects of special interest and value in the study and practice of medicine. The illustrations will be found of great assistance.

Frederic P. Gorham, A. M., *Brown University.*

"I am greatly pleased with it. Perhaps the highest praise which I can give the book is to say that it more nearly approaches the course I am now giving in general biology than any other work."

Stengel & Fox's Pathology

Pathology. By ALFRED STENGEL, M. D., Sc. D., Professor of Medicine, University of Pennsylvania; and HERBERT FOX, M. D., Director of the Pepper Laboratories of Clinical Medicine, University of Pennsylvania. Octavo of 1045 pages, with 468 text-illustrations, many in colors, and 15 colored plates. Cloth, $6.00 net; Half Morocco, $7.50 net.

NEW (6th) EDITION, REWRITTEN

This new (6th) edition is virtually a new work. It has been rewritten throughout, reset in new type, and a larger type page used. New matter equivalent to 175 pages has been added and some 75 new illustrations, many of them in colors. The work is a handsome volume of over 1000 pages. In the first portions, devoted to general pathology, the sections on inflammation, retrogressive processes, disorders of nutrition and metabolism, general etiology, and diseases due to bacteria were wholly rewritten or very largely recast. A new section on transmissible diseases was added; the terata were included, with a synoptical chapter on teratology. The glands of internal secretion were given a separate chapter, and new chapters on the pathology of eye, ear, and skin were added. **August, 1915**

Stiles' Human Physiology

(Published July, 1916)

The simplicity, the clearness with which Dr. Stiles presents this difficult subject makes the work decidedly valuable as a text-book for High Schools and General Colleges. The text is illustrated in Dr. Stiles' usual striking way.

12mo of 405 pages, illustrated. By PERCY GOLDTHWAIT STILES, Assistant Professor of Physiology at Harvard University. Cloth, $1.50 net.

Stiles' Nutritional Physiology

SECOND EDITION—published November, 1915

Prof. Stiles' work opens with a brief but adequate presentation of the physiology of free-living cells and leads up to the more complex function in man. It discusses the rôle each organ, each secretion plays in the physiology of nutrition—in the transformation of energy.

12mo of 208 pages, illustrated. By PERCY G. STILES, Harvard University. Cloth, $1.25 net.

Stiles' The Nervous System

This new book is really a physiology and anatomy of the nervous system, emphasizing the means of conserving nervous energy. You get chapters on the minute structure, reflexes, afferent nervous system, neuromuscular system and fatigue, autonomic system, the cerebrum and human development, emotion, sleep, dreams, causes of nervous impairment, neurasthenia, hygiene. **November, 1914**

12mo of 230 pages, illustrated. By PERCY GOLDTHWAIT STILES, Harvard University. Cloth, $1.25 net.

Jordan's General Bacteriology

A Text-Book of General Bacteriology. By EDWIN O. JORDAN, PH.D., Professor of Bacteriology in the University of Chicago and in Rush Medical College. Octavo of 667 pages, illustrated. Cloth, $3.25 net.

FIFTH EDITION—published September, 1916

Professor Jordan's work embraces the entire field of bacteriology, the non-pathogenic as well as the pathogenic bacteria being considered, giving greater emphasis, of course, to the latter. There are extensive chapters on methods of studying bacteria, including staining, biochemical tests, cultures, etc.; on the development and composition of bacteria; on enzymes and fermentation-products; on the bacterial production of pigment, acid and alkali; and on ptomains and toxins. Especially complete is the presentation of the serum treatment of gonorrhea, diphtheria, dysentery, and tetanus. The relation of bovine to human tuberculosis and the ocular tuberculin reaction receive extensive consideration.

This work will also appeal to academic and scientific students. It contains chapters on the bacteriology of plants, milk and milk-products, air, agriculture, water, food preservatives, the processes of leather tanning, tobacco curing, and vinegar making; the relation of bacteriology to household administration and to sanitary engineering, etc.

Prof. Severance Burrage, *Associate Professor of Sanitary Science, Purdue University.*

"I am much impressed with the completeness and accuracy of the book. It certainly covers the ground more completely than any other American book that I have seen."

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Anatomists, surgeons, medical men of all departments will find a great deal of real value. To those who are called upon to perform postmortem examinations this new book is indispensable because it gives them the new technic, the new interpretation of findings, the last word on the subject. **November, 1915**

Wells' Chemical Pathology

(Published March, 1914)

Chemical Pathology.—Being a Discussion of General Pathology from the Standpoint of the Chemical Processes Involved. By H. GIDEON WELLS, PH. D., M. D., Assistant Professor of Pathology in the University of Chicago. Octavo of 616 pages. Cloth, $3.25 net.

SECOND EDITION

Dr. Wells' work is written for the physician, for those engaged in research in pathology and physiologic chemistry, and for the medical student. In the introductory chapter are discussed the chemistry and physics of the animal cell, giving the essential facts of ionization, diffusion, osmotic pressure, etc., and the relation of these facts to cellular activities. Special chapters are devoted to *Diabetes* and to *Uric-acid Metabolism and Gout.*

Wm. H. Welch, M. D.

Professor of Pathology, Johns Hopkins University.

"The work fills a real need in the English literature of a very important subject, and I shall be glad to recommend it to my students."

Lusk's Elements of Nutrition

Elements of the Science of Nutrition. By GRAHAM LUSK, PH. D., Professor of Physiology at Cornell Medical School. Octavo volume of 641 pages. Cloth, $4.50 net. Published July, 1917

THIRD EDITION—TRANSLATED INTO GERMAN

Prof. Lusk presents the scientific foundations upon which rests our knowledge of nutrition and metabolism, both in health and in disease. There are special chapters on the metabolism of diabetes and fever, and on purin metabolism. The work will also prove valuable to students of *animal dietetics* at agricultural stations.

Lewellys F. Barker, M. D.

Professor of the Principles and Practice of Medicine, Johns Hopkins University.

"I shall recommend it highly to my students. It is a comfort to have such a discussion of the subject in English."

Daugherty's Economic Zoölogy

Economic Zoölogy. By L. S. DAUGHERTY, M. S., Ph. D., Professor of Zoölogy, State Normal School, Kirksville, Mo., and M. C. DAUGHERTY, author with Jackson of "Agriculture Through the Laboratory and School Garden." Part I: *Field and Laboratory Guide.* 12mo of 237 pages, interleaved. Cloth, $1.25 net. Part II: *Principles.* 12mo of 406 pages, illustrated. Cloth, $2.00 net. Published October, 1912

ILLUSTRATED

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Part I—the *Field and Laboratory Guide*—is designed for practical instruction in the field and laboratory. To enhance its value for this purpose blank pages are inserted for notes.

Drew's Invertebrate Zoölogy

A Laboratory Manual of Invertebrate Zoology. By GILMAN A. DREW, PH. D., Assistant Director at Marine Biological Laboratory, Woods Hole, Mass. With the aid of Former and Present Members of the Zoölogical Staff of Instructors. 12mo of 213 pages. July, 1913 Cloth, $1.25 net.

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The subject is presented in a logical way, and the type method of study has been followed, as this method has been the prevailing one for many years.

Prof. Allison A. Smyth, Jr., Virginia Polytechnic Institute
"I think it is the best laboratory manual of zoölogy I have yet seen. The large number of forms dealt with makes the work applicable to almost any locality."

Norris' Cardiac Pathology

Studies in Cardiac Pathology. By GEORGE W. NORRIS, M.D., Associate in Medicine at the University of Pennsylvania. Large octavo of 235 pages, 85 illustrations. Cloth, $5.00 net. **Published April, 1911**

SUPERB ILLUSTRATIONS

The illustrations are superb. Each illustration is accompanied by a detailed description; besides, there is ample letter press supplementing the pictures.

Boston Medical and Surgical Journal

"The illustrations are arranged in such a way as to illustrate all the common and many of the rare cardiac lesions, and the accompanying descriptive text constitutes a fairly continuous didactic treatise."

McConnell's Pathology

A Manual of Pathology. By GUTHRIE MCCONNELL, M. D., Assistant Surgeon, Medical Reserve Corps, U. S. Navy. 12mo of 600 pages, with 187 illustrations. Cloth, $2.75 net. **Published September, 1915**

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Dr. McConnell has discussed his subject with a clearness and precision of style that make the work of great assistance to both student and practitioner. The illustrations have been introduced for their practical value.

New York State Journal of Medicine

"The book treats the subject of pathology with a thoroughness lacking in many works of greater pretension. The illustrations—many of them original—are profuse and of exceptional excellence."

McConnell's Pathology and Bacteriology

For Dental Students

Pathology and Bacteriology for Dental Students. By GUTHRIE MCCONNELL, M. D., Assistant Surgeon, Medical Reserve Corps, U. S. N. 12mo of 309 pages, illustrated. Cloth, $2.25 net. **Published March, 1915**

ILLUSTRATED

This work is written expressly for dentists and dental students, emphasizing throughout the application of pathology and bacteriology in dental study and practice. There are chapters on disorders of metabolism and circulation; retrogressive processes, cell division inflammation and regeneration, granulomas, progressive processes, tumors, special mouth pathology, sterilization and disinfection, bacteriologic methods, specific micro-organisms, infection and immunity, and laboratory technic.

Bosanquet on Spirochaetes

(Published May, 1911)

Spirochætes: A Review of Recent Work, with Some Original Observations. By W. CECIL BOSANQUET, M.D., Fellow of the Royal College of Physicians, London. Octavo of 152 pages, illustrated. $2.50 net.

ILLUSTRATED

This is a complete and authoritative monograph on the spirochætes, giving morphology, pathogenesis, classification, staining, etc. Pseudospirochætes are also considered, and the entire text well illustrated. The high standing of Dr. Bosanquet in this field of study makes this new work particularly valuable.

Sobotta and Huber's Human Histology

Atlas and Epitome of Human Histology. By PRIVATDOCENT DR. J. SOBOTTA, of Wurzburg. Edited, with additions, by G. CARL HUBER, M. D., Professor of Histology and Embryology in the University of Michigan, Ann Arbor. With 214 colored figures on 80 plates, 68 text-illustrations, and 248 pages of text. Cloth, $4.50 net. *In Saunders' Hand-Atlas Series.* **Published 1903**

Boston Medical and Surgical Journal

"In color and proportion they are characterized by gratifying accuracy and lithographic beauty."

Lehmann, Neumann, *and* Weaver's Bacteriology

Atlas and Epitome of Bacteriology: INCLUDING A TEXT-BOOK OF SPECIAL BACTERIOLOGIC DIAGNOSIS. By PROF. DR. K. B. LEHMANN and DR. R. O. NEUMANN, of Würzburg. *From the Second Revised and Enlarged German Edition.* Edited, with additions, by G. H. WEAVER, M. D., Assistant Professor of Pathology and Bacteriology, Rush Medical College, Chicago. In two parts. Part I.—632 colored figures on 69 lithographic plates. Part II.—511 pages of text, illustrated. Per part: Cloth, $2.50 net. *In Saunders' Hand-Atlas Series.* **Published 1901**

Ball's Bacteriology

Seventh Edition, Revised

ESSENTIALS OF BACTERIOLOGY: being a concise and systematic introduction to the Study of Micro-organisms. By M. V. BALL, M. D., Late Bacteriologist to St. Agnes' Hospital, Philadelphia. 12mo of 289 pages, with 135 illustrations, some in colors. Cloth, $1.25 net. *In Saunders' Question-Compend Series.*

Published December, 1913

"The technic with regard to media, staining, mounting, and the like is culled from the latest authoritative works."—*The Medical Times*, New York.

Budgett's Physiology

Fourth Edition

ESSENTIALS OF PHYSIOLOGY. Prepared especially for Students of Medicine, and arranged with questions following each chapter. By SIDNEY P. BUDGETT, M. D., formerly Professor of Physiology, Washington University, St. Louis. Revised by HAROLD E. B. PARDEE, M. D., Instructor in Physiology, Columbia University (College of Physicians and Surgeons), New York. 12mo volume of 206 pages, illustrated. Cloth, $1.25 net. *Saunders' Question-Compend Series.*

Published November, 1915

"He has an excellent conception of his subject. . . It is one of the most satisfactory books of this class"—*University of Pennsylvania Medical Bulletin.*

Leroy's Histology

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ESSENTIALS OF HISTOLOGY. By LOUIS LEROY, M. D., Professor of Histology and Pathology, Vanderbilt University, Nashville, Tennessee. 12mo, 263 pages, with 92 original illustrations. Cloth, $1.25 net. *In Saunders' Question-Compend Series.*

Published August, 1909

"The work in its present form stands as a model of what a student's aid should be; and we unhesitatingly say that the practitioner as well would find a glance through the book of lasting benefit."—*The Medical World*, Philadelphia.

Durck and Hektoen's General Pathologic Histology

ATLAS AND EPITOME OF GENERAL PATHOLOGIC HISTOLOGY. By PR. DR. H. DURCK, of Munich. Edited, with additions, by L. HEKTOEN, M. D., Chicago. 353 pages, illustrated. Cloth, $5.00 net. *Saunders' Hand-Atlases.*

Published October, 1904

American Pocket Medical Dictionary

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Published April, 1915

Bohm, Davidoff, and Huber's Histology

Second Edition

A TEXT-BOOK OF HUMAN HISTOLOGY. By DR. A. A. BOHM and DR. M. VON DAVIDOFF, of Munich, and G. CARL HUBER, M.D., U. of Penna. Octavo of 528 pages, 361 illustrations. Cloth, $3.50 net. **August, 1904**